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The Present Status of Influenza Virus

RONALD HARE

Causes of Maternal Deaths in Manitoba

NOEL R. RAWSON

Abstracts of Papers from the Christmas Meeting of the Laboratory Section

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The Present Status of Influenza Virus*

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WE all know what we mean by influenza, but when we come to put down a definition on paper, it will probably be no more exact or informative and certainly in much less beautiful language than that used by Willis in 1658 when he wrote:

"As to the symptoms joyned with this disease; a feaverish intemperature and whatsoever belongs to this, the heat of the praecordia, thirst, a spontaneous weariness, pain in the head loyns and limbs were induced from the blood growing hot, and not sufficiently eventilated.

"The prognostick of this disease, concerning private persons, is, for the most part, easie, that one may deliver the event from the first assault; for if this sickness be excited in a strong body, and healthful before, and that the feaverish distemper be moderate, and without any grievous and horrid symptom, the business is free from danger; and the distemper is to be accounted but of light moment; as that commonly is of catching cold; neither needs a physician be consulted, nor remedies, unless trivial and ordinary be administered. But if this distemper happens in a weak and sickly body with an evil provision . . . the event of the disease is much to be suspected, and often terminates in death."

It will no doubt be objected that this description of the disease is unconvincing and inadequate, for, after all, "feaverish intemperature" and "pain in the head loyns and limbs" are symptoms common to quite a number of other diseases; and "spontaneous weariness" is only too common in the very healthy. Nor is the statement that "the prognostick of this disease . . . is for the most part easie" of any assistance, for acute infections of this sort usually terminate thus. But consideration will show that except for the fact that the disease is usually epidemic, taking a continent or two in its stride, there is in fact no "grievous and horrid symptom" by which to distinguish the disease from a host of imitators.

**Presented at a meeting of the Academy of Medicine, Toronto, Section of Preventive Medicine and Hygiene, November 28, 1940; and at the ninth Christmas meeting of the Laboratory Section, Canadian Public Health Association, Toronto, December 16, 1940.*

This is emphasized in no uncertain manner when we come to discuss the various agents which from time to time have produced illnesses of an influenza-like nature.

AGENTS WHICH MAY CAUSE INFLUENZA OR INFLUENZA-LIKE SYNDROMES

The first of these I shall mention only to condemn. This is the organism isolated in the epidemic of 1898-92 by Pfeiffer and which usually bears his name. Until 1918 it was generally believed than influenza was due to this organism, but in the pandemic of that year, it appeared so infrequently that its claim to be a cause of the disease could not be substantiated. Time has not brought about its rehabilitation

In the year 1933, Smith, Andrewes and Laidlaw isolated a virus from cases of influenza by the instillation of washings from the nasopharynx of clinical cases into the nares of ferrets. Since that year many similar strains of similar virus have been isolated by the same method in many parts of the world.

In many respects the disease in the ferret resembles that in human beings. After an incubation period of forty-eight or seventy-two hours there is a sudden rise in temperature which persists for twenty-four hours or more. At the same time the animal manifests signs of respiratory tract involvement in that there is a mucous or muco-purulent secretion from the nose, the animal goes off its food, becomes listless, and if disturbed is highly irritable. Recovery usually sets in after a day or two. Lung involvement with recently isolated virus almost never occurs, and death is likewise unknown. With passage virus, on the other hand, pneumonia characterized by varying degrees of bluish-red consolidation of the lungs occurs, and death may occur with extensive pulmonary consolidation. The principal lesion is, however, in the mucous membrane of the turbinates which undergoes necrosis with desquamation of the superficial cells and an inflammatory reaction in the submucosa. Repair begins relatively early—on the fourth day after infection, and by the twenty-first day the epithelium is almost normal again.

Other laboratory animals are on the whole much less susceptible than the ferret, but if ferret passage virus be employed, it is possible to produce, by nasal instillation in the mouse, varying degrees of pulmonary consolidation, and a great deal of research on this virus has actually been carried out in the mouse.

There is a great deal of evidence that this virus is the cause of many cases of influenza. In the first place, there is the classical instance of direct infection of a human being from a sick ferret. This occurred in the early summer of 1936, when influenza was completely absent in England. A ferret in the acute stage of the disease sneezed at Dr. Stuart-Harris, and within forty-eight hours he came down with a typical attack of influenza and the same virus was isolated from his nasopharynx. In the second place, a group of Russian workers persuaded no less than 72 volunteers to have virus instilled into the nostrils, and 20 per cent actually went down with typical clinical influenza. In the third place, serum taken from cases during convalescence will neutralize the virus whereas that taken during the acute phase will not or only to a much lower titre. If there had been doubts, the claims of this virus became almost unassailable in 1937, for

in the early months of that year there was a wide-spread epidemic in Europe and America. Virus was isolated in the ferret from a high proportion of the cases, and the appearance of virus-neutralizing antibodies in the serum was also detected in almost 100 per cent of the cases. At the end of 1937, therefore, it seemed certain that this virus was the principal cause of epidemic influenza.

There was, however, even at that time some evidence that this was not the whole of the story. In 1935, the London workers had been unable to isolate this virus from typical cases at Woolwich during January and February of that year, whereas in February and March they were able to isolate it with ease from cases at Shorncliffe. Then in 1936, Francis studied an epidemic in California in which the clinical picture was indistinguishable from that of other epidemics in which virus could be isolated; but despite every care, no illness indicative of the presence of a virus could be produced in the ferret, nor could an increase in antibody for the known strains of virus be detected in the serum of the patients. I will refer to this epidemic presently.

Then came the year 1939 in the early months of which there was again a wide-spread epidemic in Europe and America. Clinically and epidemiologically it resembled closely that of 1937 which, as I mentioned just recently, yielded so rich a harvest of virus. But instead of isolating virus from the great majority of the patients, as had been the case in 1937, most workers were only able to isolate very few strains in 1939. To make matters worse, there was absolutely no clinical difference between cases in which the virus could and in those in which it could not be isolated.

It therefore became apparent that there must be more than one etiological agent responsible for clinical influenza but direct evidence of this has only recently been forthcoming.

In the early months of 1940 there occurred a limited epidemic at a convalescent home outside New York. Virus could not be detected in these cases by the ordinary methods, and the sera of convalescents exhibited no increase in antibody level for the known strains of virus. By prolonged ferret passage, however, Francis was able to show that there was a virus in the nasopharynx of these cases which would eventually produce pulmonary lesions in ferrets. The illness produced in the ferret was so slight and so fleeting that it is doubtful whether many workers would have recognized it. Nevertheless a virus was present which could be transferred to mice, giving them pneumonia from which a high proportion of them died. This new virus was shown to be immunologically quite unlike previously known strains of influenza virus. It was in fact a new virus. That it was the cause of the outbreak in which it was isolated was shown by the fact that the convalescent serum of the patients neutralized this virus but of course had no action on any strains previously known. Subsequent work has shown that this virus was probably responsible for the 1936 outbreak in California to which I have already referred, since sera from convalescents in this outbreak have now been shown to possess antibodies for this new virus. For the same reason, it is also probable that this virus was the etiological agent responsible for some of the cases in the 1939 outbreak.

Thus we have clear evidence that there are at least two viruses acting as etiological agents in cases of influenza. Names therefore become desirable for *Nomina si nescis, perit et cognitio rerum.* To this end, an agreement has been reached to call the first of these viruses and the disease it causes by the soulless if innocuous term "influenza A". This is the virus which was first discovered and which was so predominant in 1937. In conformity, the second virus has been named "influenza B" and any further candidates for this alphabetical galaxy will be named accordingly.

There are indeed extra galactic agents already waiting to be admitted within the fold. Reiman and Havens recently described an epidemic of what they called pneumonitis which occurred in Philadelphia in the early months of 1939 from which a virus could not be isolated in the ferret or serological evidence obtained that influenza A virus took any part in the infection. But recently Weir and Horsfall have been able to isolate a virus from similar cases by use of the Jamaican mongoose. Convalescent serum from the cases neutralized this virus whereas that of cases in the acute phase had no such action. No other animal is known to be susceptible and the difficulties encountered in working with the mongoose, which is quite devoid of any finer feelings, are considerable. Nevertheless, the type of infection from which the virus was derived is being increasingly recognized and it is possible that a third virus is responsible for cases of an influenza-like nature.

But other viruses may, on occasion, cause outbreaks of disease which resemble clinical influenza so closely that it is almost impossible to make a differential diagnosis. Thus, the virus of African Rift Valley Fever, and that of lymphocytic choriomeningitis, have behaved in this way. Bacteria too may cause infections resembling influenza very closely. Haemolytic streptococci, for instance, and possibly pneumococci may produce an infection of an influenza-like nature. The early stages of many of the acute exanthemata may be indistinguishable from influenza and in the *formes frustes* of these infections the diagnosis may never be completely established. And, lastly, rickettsiae make confusion well-confounded, for a group of workers at the National Institute in Washington have recently described a small epidemic in which the symptoms were influenzal in nature although there was rather a longer febrile period than in influenza proper. By passage through guinea pigs, rickettsiae were isolated identical with those of Australian Q fever.

Thus a number of different agents are known, all of which have at various times given rise to infections of an influenza-like nature in human beings, and there are probably many more as yet quite unknown.

Now, I want to stress this point because I think that we must realize that influenza is not a single disease but a whole group of diseases all having a more or less common symptomatology and pathology but due to a variety of different agents. I can only compare it to the different forms of gastro-intestinal infection in which a variety of different microbes produce a very similar train of symptoms. At present it is not possible to diagnose these different forms of influenza clinically, but I have no doubt that we shall eventually be able to do so.

For the remainder of my time I shall review what is known about immunity to influenza A virus and the possibilities of immunization against it.

IMMUNITY

The host reacts to infection by the production of virus-neutralizing and complement-fixing antibodies. These antibodies appear relatively quickly during the infection and there is evidence that the titre very quickly dies away. This may possibly explain the occurrence of several attacks of the disease in a lifetime. There is also evidence that contacts during an epidemic may undergo subclinical infection, as shown by an increase in the titre of these antibodies. This lack of persistence of immunity to clinical infection may render the production of permanent solid immunity by immunization procedures very difficult, if not impossible.

IMMUNIZATION

Active Immunity

The virus is strictly pneumotropic and will not infect if injected subcutaneously or intraperitoneally. Nevertheless, virus injected intraperitoneally into a susceptible animal such as the mouse will produce a marked degree of solid immunity. This is a very simple experiment to carry out and has been confirmed many times. For a time an almost insuperable difficulty in the use of such methods for the immunization of human beings lay in the fact that the immunity produced appeared to be to a large extent strain specific, but more recent work has shown that this is mostly a question of dosage, and provided sufficient virus be used for the immunizing injection, widely valent immunity for other strains of influenza A virus can be produced. Naturally, the use of living virus in immunization of a susceptible population such as man is not in the least desirable and other methods have been suggested for inactivating the virus without rendering it non-antigenic. Formalin appears to act very well but virus suspensions inactivated by heat have also been employed.

While it is possible to produce a high degree of solid immunity in the mouse, it is very difficult, if not impossible, to produce the same degree of immunity in the ferret, and there is also some evidence that this applies to man as well. In the early months of 1940, however, a fortunate accident at the Rockefeller Institute suggested new methods for the fabrication of vaccines of much higher potency and, therefore, more likely to be of value for human beings. It was discovered that a vaccine made by the formalization of a lung from a ferret infected with both influenza virus and the virus of canine distemper would produce in other ferrets complete solid immunity to influenza whereas ferret lungs containing influenza virus alone were practically without effect. This vaccine obviously might be of use for the immunization of human beings. As ferret lung tissue is not a desirable substance to use for the immunization of human beings, experiments were therefore initiated which led to the production of such a vaccine by simultaneous growth of influenza and distemper viruses in the developing egg. Vaccines of this type are now being produced in large quantity. These vaccines can be shown

to cause an increase in the antibody level of human beings far in excess of that achieved by any other method, and it is possible that a high proportion of inoculated individuals may be rendered immune to all types of influenza A by these injections. It is, as yet, much too early to say just what the value of this vaccine may be, but it is at least an advance.

Passive Immunity

In the 1918 epidemic a number of workers reported that human convalescent serum was of value in the treatment of the more severe types of influenza. In 1933 I obtained similar results in the influenza epidemic of that year. Attempts to produce an immune serum by the injection of virus into the horse have not proved very successful, the serum produced being relatively low in titre and almost devoid of protective powers. The serum of ferrets convalescent from the disease is, however, much more powerful and in the mouse it is possible to show that such serum, given intraperitoneally, can protect the animal against many lethal doses especially if injected before the virus, and to exert some curative action if given twenty-four or forty-eight hours after the virus. It is, however, practically impossible to show that such sera are of any value whatever in the experimental infection of the ferret. No attempts have yet been made to employ these sera in the treatment of the disease in human beings.

CONCLUSION

I think I have said enough to make you appreciate that, although we know a great deal more about influenza than we did ten years ago, we still have a very long road to travel, and we might as well recognize that we are still unable to control an epidemic by any other method than invocations to St. Sebastian, the patron saint of epidemics.

Nevertheless, influenza is of extreme importance just now, for we have real epidemiological problems in the congregation of highly susceptible young men in military camps, the evacuation of whole populations of noncombatants, and the herding together of civilians in cold, ill-ventilated air-raid shelters, all of which provide ideal conditions for the spread of epidemic disease. Fortunately, this has not yet occurred to any extent. But when the sirens moan, or as Vergil put it in one of the finest and most descriptive lines in any language,

"At tuba terribilem sonitum procul aere canoro
Increpuit"

and the inhabitants of Europe go below to their shelters they may do well to recall with Laertes

"Contagious blastments are most imminent,
Be wary then."

Maternal Deaths in Manitoba*

THE FINDINGS OF THE PREGNANCY SURVEY CONDUCTED BY THE DEPARTMENT OF HEALTH AND PUBLIC WELFARE OF MANITOBA FROM MAY 1, 1938 TO APRIL 30, 1940

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IN face of a comparatively high rate for maternal deaths, and with no sign of a fall in this rate, organized medicine throughout the world has, since the late twenties, given special interest to the condition of child-bearing. In 1936 the Dominion Council of Health, the advisory body to the Federal Department of Pensions and National Health, proposed to establish demonstration areas in which the advantage of new methods could be shown and their value tested. It was decided that information should first be obtained on the present conduct of pregnancy and of childbirth. For the formulation of the scheme a special committee was appointed under the chairmanship of Doctor J. T. Phair, Chief Medical Officer of Health for the Province of Ontario.

Some area had to be chosen in which a survey could be made profitably, an area as representative as possible of the Dominion as a whole and with the machinery adaptable to the carrying out of the program. For many years the problem of maternal mortality had been a matter of keen study by the profession of Manitoba. On the inception of the Department of Health and Public Welfare in 1928, Dr. E. W. Montgomery, its first minister, established an inquiry into the causes of maternal deaths which is still being pursued. In this, each physician who reports the death of a woman which is associated with pregnancy or childbirth is asked to complete a special form giving information on the various factors that may have had an influence on the fatal issue. Therefore, Manitoba was chosen as the area for the survey into the management of pregnancy and childbirth, with special reference to maternal welfare. The period for the survey was May 1, 1938 to April 30, 1940. The Dominion Government provided a doctor and a nurse to conduct the collection of the returns; the Provincial Government provided a nurse and necessary office premises; and the Rockefeller Foundation gave a grant of $16,000.00 to cover the fees to the medical practitioners for their reports and other expenses. Grants were also made by the Canadian Medical Association and the College of Physicians and Surgeons of Manitoba. The responsibility for the direct administration of the survey was placed with the Department of Health and Public Welfare, with the Maternal Welfare Committee of the Canadian and Manitoba Medical associations as advisers.

The card to be completed by the practitioner at the first interview, and

*Presented before a joint session of the Canadian Public Health Association (twenty-ninth annual meeting) and the Manitoba Medical Association, Winnipeg, September 16, 1940.

the pregnancy record form to be kept by him throughout the pregnancy, delivery, and puerperium, were drawn up by this committee. The committee was responsible also for drawing up the list for the coding of the various points enumerated on the card, the pregnancy record, and the delivery report. The list comprises eighty-nine headings of twelve items each. After a preliminary examination of the first and second months of the survey, conducted this year, the code list was revised.

PREGNANCY SURVEY—PROVINCE OF MANITOBA

MAY 1, 1938—APRIL 30, 1940

REPORT ON MATERNAL DEATHS

FORM I

Confidential FIRST NOTIFICATION OF PREGNANCY

Serial No..... Date.....

PATIENT'S NAME.....Address.....

Age.....Usual Weight.....Height Without Shoes.....

Racial Origin of Patient's (Father.....Living.....Dead.....Cause.....

(Mother.....Living.....Dead.....Cause.....

Racial Origin of Child's Father.....Living.....Dead.....Cause.....

Social Status—Comfortable.....Moderate.....Poor.....Destitute.....

Marital Status—Single.....Married.....Widow.....

First day of last Menstruation.....Expected Date.....

Previous Health of Patient (Special reference to previous pregnancies, deliveries, toxaemias, Haemorrhage, etc.).....

.....

Signature.....

Address.....

If anything special to note use back of card.

This form if placed in an envelope, marked "Dominion Statistics—Free, penalty for improper use $300," and properly addressed will pass through the mail "FREE"

During the period of the survey there were recorded 27,965 births, of which 23,422 were attended by a doctor. The number of pregnancy records received was 22,187; and of these, 1,220 were abortions. Thus records were obtained for 89.5 per cent of the births receiving medical attendance.

During the same period there were recorded 122 deaths associated with pregnancy. Of these, eighty-nine were attributed to puerperal causes and thirty-three to other causes, giving a maternal death rate of 3.26 per 1000 live births. It must be noted that this survey occurred at a time when the maternal death rate throughout the civilized world was low. Of the eighty-nine deaths due to puerperal causes twenty-five were associated with abortions or births under twenty-eight weeks' gestation, and two others were deaths of mothers in which no birth occurred. As has been frequently claimed, the rating of maternal deaths on live births appears to be irrational, but as there is no knowledge of the number of pregnancies that end in abortion and the reporting of still-births is not universally demanded the number of live births remains the only reliable denominator.

PREGNANCY SURVEY, PROVINCE OF MANITOBA

MAY 1, 1938-APRIL 30, 1940

REPORT OF MATERNAL DEATHS

FORM 2

CONFIDENTIAL

Serial No..... **PREGNANCY RECORD** Expected Date.....

1

NAME ..... Address.....

Number of Previous—Live births.....Still births (over 28 weeks).....Abortions (under 28 weeks).....

PRENATAL PERIOD

2

Care given by (Yes or No) Doctor.....Nurse.....None.....

Calendar Months at which the following occurred	2	3	4	5	6	7	8		9	
							1st ½	2nd ½	1st ½	2nd ½
1. Vomiting										
2. Headache										
3. Oedema										
4. Visual Disturbance										
5. Epigastric Pain										
6. Blood Pressure (Systolic										
(Diastolic										
7. Urinalysis (if abnormal give details under summary)										
8. Weight										
9. Haemoglobin										
10. Presentation										
11. Position										
12. Foetal Heart Sounds										
13. Height Fundus above Symphysis (Centimeters)										
14. Wassermann										
15. Haemorrhage										

Cause of Haemorrhage.....

3 PHYSICAL EXAMINATION	**4** FOCI OF INFECTION	**5** PELVIC MEASUREMENT (in Centimeters)
1. Heart.....	1. Naso Pharynx.....	1. Interspinous.....
2. Lungs.....	2. Teeth.....	2. Intercristal.....
3. Throat.....	3. Chest.....	3. Ext. Conjugate.....
4. Teeth.....	4. Urinary tract.....	4. Transverse Outlet.....
5. Breasts.....	5. Pelvic Viscera.....	5. Internal Conjugate.....
6. G. U. Tract.....	6. Vulvovaginal.....	6. Is Pelvis Abnormal (Yes or No)...
7. Evidence Toxaemia (Date).....	7. Cervix.....	If yes, state abnormality.....
8. Quickening (date).....	8. Smear.....	.....
9. Other.....	9. Other.....	.....

Associate Conditions-Specify.....

Admitted to Hospital for Complications (Reasons).....Date.....

SUMMARY Sections 2 to 5.....

.....

.....

.....

.....

.....

.....

6 NOTE—In cases of ABORTION or MISCARRIAGE please complete the items above which are applicable, and answer the following:

1. Period of Gestation (Weeks).....2. Cause of Onset.....

3. Method of Treatment.....4. If Patient dies, cause.....

.....

Div. Child & Maternal Hygiene Dept. of P. & N.H. 1254-8-39 Blog 382

This form if placed in an envelope, marked "Dominion Statistics—Free, penalty for improper use $300," and properly addressed will pass through the mail "FREE"

PREGNANCY SURVEY, PROVINCE OF MANITOBA

MAY 1, 1938-APRIL 30, 1940

REPORT OF MATERNAL DEATHS

FORM 2 (Reverse)

Serial No.....

DELIVERY

TIME OF DELIVERY—Year.....Month.....Day.....Hour.....

Place of Delivery.....Attendant.....

7 TECHNIQUE (Check items used)

(a) Patient—Shaved.....Local Antiseptics.....Sterile Drapes.....

(b) Attendant—Caps.....Gown.....Mask.....Gloves.....Street Clothes.....White Clothes.....

8

PRESENTATION.....

Position.....

Examinations—Vaginal (Number).....

Rectal (Number).....

Foetal Heart Rate—1st Stage.....

2nd Stage.....

9

SEDATIVES USED.....

Amount.....

Hour given.....

Results.....

10 LABOUR	Duration, Hours	Anaesthetic	Pituitary Extract	Ergot Preparation
Natural.....	1st Stage.....	Type.....	Type.....	Type.....
Induced.....	2nd "	Given by.....	Amount.....	Amount.....
Method.....	3rd "		Indication.....	Indication.....

11 OPERATIVE DELIVERY	**12** PLACENTA	**13** COMPLICATIONS
Forceps, High.....Mid.....Low.....	Expelled.....	Lacerations—Cervix.....Vagina.....
Version.....Extraction.....	Expressed.....	Perineum 1st, 2nd, 3rd Degree.....
Caesarean—High.....Low.....	Manual Removal.....	Repair—Silkworm.....Catgut.....
Embryotomy.....	Incomplete.....	Haemorrhage.....
Episiotomy.....		Other Complications.....
Other Manipulations.....		

REMARKS: (Give in detail particulars of complications in Sections 10 to 13—Especially Haemorrhage).....

14

FOETUS—Sex.....Weight.....lbs.....ozs. Period Gestation, Weeks.....

Live Birth—Normal.....Abnormal (Physical or Mental).....

Dead Birth—Cause.....Before Labour.....During Labour.....Macerated.....

15 PUERPERIUM—Normal.....Febrile.....Cause.....Date of Onset.....

Course.....

Other Complications.....

.....Days in Bed.....

16 POST PARTUM EXAMINATION—Date.....Local Findings (Involution and condition of pelvic organs and floor).....

.....Breasts.....

Condition of patient — Post Partum.....

If Patient Dies, give Cause.....

Baby Breast Fed—No.....Yes.....Entirely.....Partially.....

Progress of Baby.....

NOTE: (1) DEAD BIRTH: A foetus after a minimum of 28 weeks gestation in which respiration does not take place after complete birth;

(2) PUERPERIUM; FEBRILE: A patient who, on two or more days during puerperium, other than the first twenty-four hours, has a temperature of 100.4 or over.

DATE.....M.D.

For eighty-nine pregnancies that terminated in death from maternal causes there were received seventy-seven pregnancy records and eighty-seven replies to the questionnaire in the maternal-mortality survey. For the other two there is available only the information given on the death certificate. Copies of the pregnancy records, replies to the maternal-mortality questionnaires, and death certificates, with all names omitted, have been carefully studied by the members of the obstetrical committee. It is the coding reached, after conference with them, which has been adopted in the present report. Since in many cases more than one factor is acting as a cause of death, the coding can, at best, be arbitrary but the present coding does represent the considered opinion of several experts.

Among the striking points shown from our examination of these records are:

1. The number of deaths among Indian mothers. Although Indians comprise but one-fiftieth of the population of Manitoba, they contribute more than one-tenth of the maternal deaths. The maternal death rate for the white race, including Half-breeds, falls to 3.1 per 1000 live births.

2. The deaths among Ukrainians were also excessive; they number one in nine of the population and their maternal deaths one in six. Of the fifteen deaths, eight were due to obortion.

3. The death rate among the Half-breeds is also very high.

4. Though the ratio of births out of wedlock to total live births is consistently less than 4 per cent, the ratio of maternal deaths associated therewith is over 5 per cent.

5. The number of deaths from abortions among residents of Winnipeg and Eastern Manitoba is, in comparison with the rest of the province, excessive. There was only one death by abortion of an unmarried woman.

6. The number of deaths among primiparae was far smaller than that recorded in other surveys.

7. There were remarkably few deaths of white women from septicaemia.

8. Of eighty-nine women who died of puerperal causes, ten received no medical attendance and eleven were not seen by the doctor until after the birth of the child. The death rate among white women receiving medical attendance is less than it is among those not so attended. On the other hand, the high death rate among Indian women, of those attended by the doctor, shows the fallacy of rates taken from selected samples. Where it is the custom for difficult cases only to be referred to the doctor, naturally the rates for those receiving medical attendance compare unfavourably with the average.

Prenatal Care

Prenatal care is reckoned in this study by the number of visits recorded. Complete prenatal care denotes eight visits or more; adequate, five to seven visits; partial, two to four visits. Admittedly this does not give a true picture of the quality or value of this prenatal care but it is as reliable and convenient a measure as could have been taken under the conditions of the survey.

Comparison of the prenatal care received by the eighty-nine women who died of puerperal causes and that of the 1935 who were reported to have been

delivered in May and June, 1938 and formed the subject of a preliminary study, shows little difference. Those who died had a slightly better record for complete and adequate care than the average.

Prenatal Care	Pregnant Women May, June, 1938		Women who died May 1, 1938 to April 30, 1940	
	Number	Per cent	Number	Per cent
Complete	120	6.4	8	9.1
Adequate	246	12.7	13	14.8
Partial	623	32.2	11	11.3
One visit	213	11.0	15	17.0
None	733	37.7	41	46.5

Abortions

Of the twenty deaths assigned to abortions, fifteen were attributed to sepsis. Of these, six were admittedly self-induced. In three cases autopsy showed death to have been caused by air embolism of the heart and brain, the women having been found dead with evidence pointing to the self-induction of abortion, one with a syringe and bowl of fluid at her side and one with a metal catheter. Profuse haemorrhage was stated on four occasions and in one case this seemed to be the sole cause of death. One woman was brought to the hospital suffering from all the signs of acute toxic hepatitis and her blood gave a strongly positive Wassermann reaction. Two deaths occurred after therapeutic curettage. Blood transfusion was given on two occasions and prontylin on two occasions. Medical attendance was received by all but the three women who were found dead. Fifteen were admitted to hospital.

Of the nine self-induced abortions four were in primiparae. Eight of the abortions were of Ukrainian women, none of Indian. Only one of the twenty was a single woman.

Haemorrhage

Death occurred from haemorrhage on sixteen occasions. On three it was due to malinsertion of the placenta and on seven to atony of the uterine muscle. Of the latter there were four cases of twins, one of an overmatured, oversized child, and one of a shoulder presentation; and for one there was no information.

According to the records, blood transfusion was administered to one patient only; saline glucose was given intravenously to three and haemostatic serum used twice. Usually the suddenness of the death or the absence of the doctor prevented adequate measures being taken. It is to be noted that eleven of these sixteen deliveries were in hospital.

Septicaemia

Septicaemia shows a remarkably low record in this series. Only fourteen women died from this cause in the two years and of these only eight had been attended by a doctor at the time of the delivery. Four of the deaths were of Indian women and of these one had been attended at the confinement by a medical man although not until prolonged and unsuccessful attempts at delivery had been made by an Indian midwife. Three of those dying from this cause had had a caesarean section performed, one of them being an Indian

woman already mentioned; a second, after an unsuccessful attempt to deliver interlocked twins with forceps; and a third for a fibroid condition, hysterectomy being performed also in this case.

In four of the women dying from septicaemia delivery had been assisted manually or with instruments. There were three versions, in one of which caesarean section would have been preferred but that the patient's abdomen was densely scarred from old burns. In the fourth case there was manual removal of the placenta. The eighth woman who had been attended by a doctor had a normal labour, which was followed by repeated haemorrhage, not apparently large in extent, which her mother had attempted to stop by filling the vagina with filthy rags.

Of the unattended cases septicaemia was traced in one case to fibromyoma and in a second to retained placenta.

Toxaemia

There were eighteen deaths due to toxaemia. In two of these acute yellow atrophy of the liver was discovered at autopsy. The others were of renal or eclamptic type. Two of the deaths occurred before the completion of twenty-eight weeks' gestation, one of these being due to acute yellow atrophy in an Indian girl of nineteen years. The number of primiparae—nine out of eighteen—is striking. Five were in their second pregnancy.

Toxaemia is generally recognized as the condition above all others to benefit by prenatal care, yet during the period of the survey a higher proportion of those who died from toxaemia had received prenatal care and medical attention than of those from any other of the four main causes. Six of the eighteen had received complete or adequate care and fourteen had been attended by a doctor at the time of the labour. Preliminary symptoms of oedema, rising blood pressure, or albuminuria had been noted in all but one of the eleven who had received prenatal care. In one case there had been hypertension with a systolic blood pressure of over 200 throughout pregnancy. Another woman gave a history of chronic nephritis. In five, early symptoms were recognized two to four weeks before delivery. In three the onset of labour and death followed closely upon the recognition of the first symptoms. In one case, pregnancy, childbirth and puerperium passed happily and uneventfully until eleven days after delivery when the patient began to feel nauseated. She died on the eighteenth day; autopsy showed a haemorrhage into the substance of the liver and evidence of postpartum eclampsia.

Embolism and Sudden Death

There were six deaths due to embolism and sudden death. All the deceased had been attended by physicians at the time of the delivery. Two deliveries proceeded naturally; of these cases, one death was attributed by the attending physician to visceral recoil, the other the doctor described as psychological death. Labour was induced in four cases. In three of these pulmonary embolism was cited as cause of death; in two cases this occurred shortly after labour and in the third two weeks later. In two the labour had been induced for signs of preeclamptic toxaemia, in a third for bleeding from a

marginal placenta praevia. In the fourth case the uterus had been treated with radium for endo-metritis and, although in the forty-second week, it showed no signs of contraction or of dilatation of the rigid cervix. Labour was induced medically and also by dilatation of the cervix manually but there was little response. The labour lasted ninety-six hours, the child being removed by version.

Ectopics

There were four deaths from ectopic pregnancy. Self-induction of abortion had been attempted in two. Three of the women were seen before the rupture of the ectopic but the condition was not recognized. Laparotomy was performed in three of the cases, and death occurred shortly in two of these. The third woman did well until ten days later, when there appeared gangrene of the foot evidently due to thrombosis or embolism; she died on the thirteenth day.

Other Causes

Five deaths were directly attributed to strain and shock entailed through difficulty of delivery; in three this was due to malposition of the foetus, in one to contraction of the pelvis, and in one to obstruction by an ovarian cyst. The contracted pelvis was in a fifteen-year-old Indian girl who was brought to hospital on the fourth day of labour. Delivery was made by caesarean section. The ovarian cyst was not discovered until the onset of labour. Delivery was made by a low caesarean section but the haemorrhage from the uterine wall could not be controlled and the patient died, despite blood transfusion. One mother had a previous caesarean section which had left adhesions. During labour these ruptured and the patient showed signs of internal haemorrhage but refused to go to hospital until she was in extremis. By a post-mortem caesarean section a hydrocephalic child was delivered; this was the fourth hydrocephalic in a family of five.

No efficient cause was apparent for three of the deaths. In one woman labour was induced with quinine, as she lived at a distance from the hospital and was becoming oversized, but she died suddenly before the birth of the child. In another case the doctor described a succession of weak spells which were for some hours controlled with glucose and blood transfusion. In the third case the mother is said to have been very frail and to have lost heart. One mother was attended by an Indian midwife and history is stated by the missionary to have suggested rupture of the uterus but no details were forthcoming.

It is just eleven years since Dr. E. W. Montgomery, in addressing the Manitoba Medical Association on the inception of the maternal mortality enquiry, stated that for several years the rate of death had been ½ per cent or 5 per 1000 live births, and that there was no sign of its reduction; that the chief cause of death was puerperal sepsis, the control of this condition being a primary problem of maternal mortality, and that the death rate was higher in cases which received medical attention. He looked forward to the time when instead of seventy maternal deaths each year there would be only fifty.

He must be particularly pleased with the change in the picture presented to-day. The maternal death rate has been reduced to 3.26 per 1000 live births; excluding the Indians, over whose circumstances the Province has little or no control, to 3.1.

Puerperal sepsis is no longer the leading cause of death; instead of a toll of twenty-five deaths a year to this cause, only fourteen were attributed to it during the two years, and of these seven were Indians and Half-breeds, and six did not receive medical attention. In place of the fifty deaths a year to which he hoped the mortality might be reduced, there were only eighty-nine during the two years. The death rate among those who had been attended by a doctor is decidedly less than among those who received no attendance.

This reduction in maternal mortality, this change in the picture, redounds greatly to the credit of the medical profession of Manitoba. No doubt, the stimulus given by the surveys has been of great assistance, while the teaching of the obstetricians has been the guiding light.

Appended hereto are certain tables which amplify the text.

TABLE I

MATERNAL DEATHS BY CAUSE

MANITOBA, MAY 1, 1938–APRIL 30, 1940

	Cause of Death	No.	Rate per 10,000 Live births*	Per cent of Whole
140a	Abortion with septic conditions	9	3.3	10.0
b	Self-induced abortion (septic)	6	2.2	6.7
141a	Abortion, sepsis not mentioned	2	0.7	2.3
b	Self-induced abortion without mention of sepsis	3	1.1	3.4
142	Ectopic gestation	4	1.5	4.5
144	Puerperal haemorrhage:			
a	Placenta praevia	3	1.1	3.4
b	Other puerperal haemorrhage	13	4.8	14.6
145a	Puerperal septicaemia (not specified as due to abortion)	14	5.1	15.7
146	Puerperal albuminuria and eclampsia	16	5.9	17.9
147	Other toxaemias of pregnancy	2	0.7	2.3
148a	Thrombosis			
b	Embolism	4	1.5	4.5
c	Sudden death	3	1.3	3.4
149a	Caesarean section	1	0.4	1.1
c	Other surgical operations	3	1.3	3.4
d	Rupture of uterus	1	0.4	1.1
e	Other accidents of childbirth	3	1.3	3.4
150b	Other conditions of puerperal state: shock	2	0.7	2.3
	All causes	89	32.6	100.0

*Number of live births: 27,287.

TABLE II

MATERNAL DEATHS—MANITOBA

MAY 1ST, 1938—APRIL 30TH, 1940

Attributed to Puerperal Causes				89
Attributed to Other Causes				33
Abortions			20	
Septic (including 6 self-induced)		15		
Air embolism (self-induced)		3		
Haemorrhage		1		
Toxaemia		1		
Toxaemia			18	
Renal		16		
Hepatic		2		
Septicaemia			14	
After caesarean section		3		
After assisted labour		4		
Normal labour: unskilled interference		1		
Unattended by doctor		6		
Haemorrhage			16	
Ante partum		4		
Avoidable	1			
Placenta praevia	3			
Post partum		12		
Atonic	8			
Retained placenta	1			
Delay in clotting	1			
No information	2			
Other puerperal causes			21	
Ectopic		4		
Embolism and sudden death		7		
Difficult delivery		5		
Malposition of foetus	3			
Obstruction by tumour	1			
Contracted pelvis	1			
Intra-abdominal haemorrhage (for adhesions resulting from previous caesarean section)		1		
No efficient cause apparent		3		
Unknown (delivered by midwife)		1		
Non-puerperal causes			33	
Tuberculosis		9		
Influenza		4		
Heart disease		7		
Lobar pneumonia		3		
Tumours		2		
Appendicitis		2		
Rheumatic fever		1		
Laryngeal abscess		1		
Exophthalmic goitre		1		
Poliomyelitis		1		
Congenital heart disease		1		
Leukaemia		1		

TABLE III

MATERNAL DEATHS

BY RACE and BY MEDICAL ATTENDANCE

MANITOBA, MAY 1, 1938—APRIL 30, 1940

	Live Births	Deaths from puerperal causes		Death rates per 1,000 live births	
		Over 28 wks. gestation	All	Over 28 wks. gestation	All
White women:					
Delivery attended by doctor.	22,542	42	62	1.9	2.7
Not attended by doctor.....	3,297	10	16	3.0	4.9
No information.....		2	2		
All white women.....	25,839	54	80	2.0	3.1
Indian women:					
Delivery attended by doctor.	267	4	4	15.0	15.0
Not attended by doctor.....	1,181	4	5	3.6	4.2
All Indian women.....	1,448	8	9	5.5	6.2
All women:					
Delivery attended by doctor.	22,809	46	66	2.0	2.9
Not attended by doctor.....	4,478	14	21	3.1	4.7
Unknown.....		2	2		
All.....	27,287	62	89	2.3	3.0

Inclusion of the two unknown among those attended by a doctor would raise the maternal death rate for these among white mothers to 2.8, and among all mothers to 3.0.

TABLE IV

MEDICAL ATTENDANCE ON DECEASED MOTHERS

BY CAUSE OF DEATH

MANITOBA, MAY 1, 1938—APRIL 30, 1940

	Abortion	Haemorrhage	Toxaemia	Septicaemia	Other	All
Medical attendance						
At birth or abortion..	14	10	14	8	20	66
After birth.....	3	2	2	4		11
None.....	3	2	2	2	1	10
No information.....		2				2
Excluding "no birth", abortions and "no information"						
Doctor in attendance at birth.....		10	13	8	16	47
Called after birth....		2	1	2		5
No attendance.....		2	1	2	1	6

TABLE V

MATERNAL DEATHS

BY AGE and BY PARITY

MANITOBA, MAY 1, 1938—APRIL 30, 1940

	Abortion	Haemorrhage	Toxaemia	Septicaemia	Other	All	Rate per 1,000 L. B. (prov.)
Age							
15–19			1		1	2	1.1
20–24	5	1	1	5	1	13	1.7
25–29	5	5	6	2	7	25	3.4
30–34	5	1	7	2	6	21	3.9
35–39	4	3	2	4	2	15	4.7
40–44	1	6	1	1	3	12	11.0
45 and over					1	1	16.0
Parity							
Primiparae	6	1	9	4	6	26	2.6
2nd para	4	1	5	2	4	16	2.5
3–5	6	4	1	5	6	22	3.5
6–9	1	2	2	3	3	11	6.9
10 and over	2	6	1		2	11	15.9
No information	1	2				3	

This period seems to have been exceptionally favourable to primiparae and those of low parity and severe to those of high parity. The rates quoted above and the relative proportion of deaths by parity may be compared with the five-year period, 1933-1937.

	Rates	Relative proportion	
	1933-1937	1938-1940	1933-1937
Primiparae	5.8	29.2	36
2nd para	4.7	18.0	22.1
3–5	3.2	24.7	22.5
6–9	4.2	12.4	19.4
10 and over		12.4	
No information		3.3	

TABLE VI

MATERNAL DEATHS

BY PERIOD OF GESTATION, BY RESULTING BIRTH and BY PRENATAL CARE

MANITOBA, MAY 1, 1938—APRIL 30, 1940

Cause of Death	Abortion	Haemorrhage	Toxaemia	Septicaemia	Other	All
Period of gestation						
Under 28 weeks	20		2		4	26
28–31			3			3
32–35		2	2	2	1	7
36–39		2	6	4	2	14
40 weeks or "term"		9	4	8	9	30
Over 40 weeks		1	1		5	7
No information		2				2
Resulting birth						
Live birth						
Single, Male		3	5	4	3	15
Female		3	2	6	3	14
Twins		4	2	1	1	8
Stillbirth		4	6	3	10	23
No birth	6		2		4	12
Abortion	14		1			15
No information		2				2
Prenatal care						
Complete (more than 7 visits)			3	1	4	8
Adequate (5-7V.)		1	3	3	6	13
Partial (2-4V.)	1	2	5		3	11
One visit	6	5	3	1		15
None	13	7	4	9	8	41
No information		1				1
Operations						
Forceps						
Low		2	1	1	1	5
Mid		2			1	3
High				3		3
Post mortem					1	1
Version		1	3	1	3	8
Caesarean		1	2		2	5
Caesarean, p.m.				3	1	4
Laparotomy					3	3
Craniotomy			1			1
Manual removal of placenta	1	1	2		1	5
Curettage	2					2
Blood transfusion	2	1		1	1	5

TABLE VII

MATERNAL DEATHS

BY PLACE OF BIRTH, BY RESIDENCE and BY MARITAL STATUS

MANITOBA, MAY 1, 1938—APRIL 30, 1940

	Abortion	Haemorrhage	Toxaemia	Septicaemia	Other	All
Birth in hospital						
Greater Winnipeg	9	2	6	2	9	28
Provincial	6	9	6	5	9	35
Birth at home						
Greater Winnipeg	2			1	1	4
Cities, towns and villages	1			2		3
Rural	2	3	5	1	1	12
Unorganized		1				1
Indian Reserves		1	1	3	1	6
Residence						
Greater Winnipeg	9		4	2	8	23
Eastern	5	3	2		1	11
South Central	1	5	1	1		8
Interlake	1	1	2	1	2	7
South Western			4		4	8
West Central	1	1	1	3		6
North Western	1	1	1	3	1	7
Northern	1	1	1		2	5
Cities	9		5	2	10	26
Towns	3	1	2		6	12
Villages		3		2		5
Rural, organized	7	7	8	6	1	29
Rural, unorganized		1	1		1	3
Outside Manitoba	1	2	1			4
Indian Reserves		2	1	4	3	10
Married	19	16	17	13	19	84
Single	1		1	1	2	5

Where no birth has occurred the place of death is cited. The numbers of births occurring in hospital and at home have not yet been ascertained nor the number of births by residential area. The proportion of deliveries that have occurred in hospital agrees very closely with that in the preliminary study of the Pregnancy Survey, 70.7 as against 70.3.

The approximate populations may be a guide in considering the above table.

Greater Winnipeg	295,000	Cities	330,000
Eastern	56,000	Towns	51,000
South Central	74,000	Villages	13,500
Interlake	52,000	Rural, organized	311,000
South Western	77,000	Rural, unorganized	30,000
West Central	63,000	Indian Reserves	14,000
North Western	64,000		
Northern	32,000		

TABLE VIII

MATERNAL DEATHS

BY RACIAL ORIGIN

MANITOBA, MAY 1, 1938—APRIL 30, 1940

	Abortion	Haemorrhage	Toxaemia	Septicaemia	Other	All	Rates per 1,000 L.B.
British	6	1	8	3	11	29	2.6
English, Welsh	5	1	7		5	18	
Scottish				3	4	7	
Irish	1		1		2	4	
French	1	1			4	6	2.1
Northern European	2	1	2	1	1	7	4.8
Norwegian	1			1		2	
Icelandic	1	1	1		1	4	
Finn			1			1	
Germanic	2	5	4	2	1	14	3.1
German	1	3	4		1	9	
Belgian				1		1	
Dutch	1	2		1		4	
Hebrew					1	1	3.3
East.CentralEuropeans	9	5	2	2	1	19	3.3
Poles		2	1			3	
Ukrainians	8	3	1	2	1	15	
Austrians	1					1	
Half-breed		1	1	2		4	13.2
Indian		2	1	4	2	9	7.5

TABLE IX

MATERNAL DEATHS

BY TIME AFTER DELIVERY

MANITOBA, MAY 1, 1938—APRIL 30, 1940

	Abortion	Haemorrhage	Toxaemia	Septicaemia	Other	All
During labour	2		3		2	7
Within 12 hours		9	7		10	26
12–24 hours		1			1	2
1–3 days		4		1	1	6
4–7 days	2		2	6	1	11
8–14 days	2		1	2	1	6
15–21 days	2		1	1		4
22–28 days	4			1	1	6
Over 28 days	2		2	3		7
Before labour	4		2			6
No information	2	2				4
Ectopic					4	4

ABSTRACTS OF PAPERS

PRESENTED AT THE NINTH ANNUAL CHRISTMAS MEETING OF THE LABORATORY SECTION, CANADIAN PUBLIC HEALTH ASSOCIATION, TORONTO, DECEMBER 16-17, 1940

●

A Laboratory Procedure for Detecting and Eliminating Thermoduric Bacteria from Pasteurized Milk—V. E. GRAHAM, University of Saskatchewan, and W. H. ORME, Department of Public Health, City of Saskatoon.

THERMODURIC bacteria are sometimes the cause of high counts in pasteurized milk. In cities which enforce a numerical limit on the plate count of such milk the presence of these organisms may cause much worry to operators of pasteurizing plants. When excessive counts are found it is often necessary to discover whether thermoduric bacteria are the cause, and if they are, to find their source so that they can be eliminated from the supply. The source is usually one or more of the farms supplying milk to the plant. In most cases it is necessary to get all this information quickly.

In an outbreak of this nature a plan was evolved which was very successful. It is being outlined here in the hope that it may be found useful by others when faced with a similar problem. An outline of our procedure follows:

1. Secure a representative sample of the hot milk immediately after pasteurization. Cool this sample quickly and take it to the laboratory.

2. Transfer 10 ml. of the sample asceptically to a sterile test tube and repasteurize immediately in the laboratory.

3. Prepare a series of plates from each sample, i.e. the one taken directly from the vat and the one pasteurized in the laboratory. Several dilutions should be used. Incubate at 37°C.

4. If a number of vats are in use, this procedure should be followed with freshly pasteurized milk from each one.

If thermoduric organisms are present there will be little or no reduction in count on the plates as a result of repasteurization in the laboratory. Usually the results from these plates can be obtained in twenty-four hours. In that time the relative number of organisms on the different series of plates can be seen by the unaided eye or by examination under a binocular dissecting microscope. Accurate and detailed counts are not necessary for this purpose.

If, as frequently happens, the source of the thermoduric organisms is the milk of one or more producers, the method of laboratory pasteurization can be used to locate the offending supplies quickly. The following procedure is satisfactory.

1. Take to the plant a supply of sterile bottles large enough to sample the milk from each shipper as it is being weighed.

2. Provide for a method of sterilizing the stirring rod. Having two stirring

rods, to be used alternately, and keeping the unused one in *boiling* water when not in use, is usually sufficient.

3. With this equipment take a sample of each producer's milk as it enters the plant. Keep the samples cold until they reach the laboratory.

4. On their arrival at the laboratory shake the samples and transfer 10 ml. from each into a sterile test tube with asceptic precautions.

5. Pasteurize these tubes immediately.

6. Plate out both the raw and the pasteurized samples. In a survey of this nature it is usually sufficient to use one dilution of each sample. For the raw milk 1:1000 and for the laboratory pasteurized milk 1:100 dilutions are usually satisfactory, but these values may be changed to suit local conditions.

Our experience would indicate that with most raw milk supplies, very few colonies will appear on the 1:100 plates after laboratory pasteurization. Where thermoduric organisms are present in considerable numbers there will be very little difference in the plates from the raw and pasteurized samples. It therefore becomes relatively easy to find the offending shippers within twenty-four to forty-eight hours by following this method. Usually the elimination of the milk from a few shippers completely changes the situation from the standpoint of bacteria counts in the pasteurized milk. It is not to be inferred that the shippers of milk containing large numbers of thermoduric bacteria are necessarily careless, nor that they should be permanently eliminated as suppliers of milk for the city trade. The procedure outlined is merely useful in solving what may be a very embarrassing problem from the standpoint of the milk plant and the health department. Further work must then be done on the farm. The only serious trouble we have experienced from this group of organisms has been due to the improper treatment of milking machines. One or two visits from the inspector coupled with a few laboratory counts on the milk have been sufficient to clear up the situation to the satisfaction of everyone.

No claims are made for any originality in the methods which have been suggested. Emphasis, however, is placed upon two facts, viz., that trouble from thermoduric organisms may appear suddenly, and that the methods outlined herein will usually provide a quick solution to what may be a very embarrassing situation. The practical value of laboratory pasteurization as a tool for the milk control laboratory is particularly emphasized. Every laboratory engaged in milk control should be provided with equipment for the pasteurization of milk in test tubes.

The Application of the Evelyn Photo-electric Colorimeter to a Modification of the Kay-Graham Phosphatase Test—J. WYLLIE, Queen's University, Kingston, Ont.*

A SIMPLE modification of the Kay-Graham phosphatase test is described in which 0.02 ml. of milk is used instead of 0.5 ml. in the original method. This enables 1 ml. instead of 4.5 ml. of Folin and Ciocaltau's reagent to be used and employs a single filtration only.

**This paper will be published in full in the March issue.*

On the basis of the results from laboratory pasteurized milk samples it appears that 95 per cent of milk samples should fall within scale readings of 18 to 35 (test A) and 29 to 44 (test B).

It is suggested that this technique may be applied to detect minor errors in the pasteurizing process of commercial dairies.

Tuberculin and Its Uses—WM. D. HAY, Queen's University, Kingston, Ont.

THE problems of tuberculosis and diagnosis are discussed briefly. Tuberculosis is a disease which we must try to find before it is clinically evident. Suitable groups of individuals for study are outlined, namely: (a) all contacts with cases of tuberculosis; (b) patients with indefinite or suspicious symptoms; (c) workers exposed to industrial hazards; and (d) individuals belonging to the age groups which are most susceptible to disease.

The means of study are discussed, especially the use of tuberculin, X-ray, etc.

A study of reactions in students of secondary schools, in nurses and students in university, is given with summary of results in the latter group especially. The value of this work is two-fold, viz., firstly, case finding and secondly, it has public health educational value amongst individuals highly susceptible to training.

Papain Digest Media and Standardization of Media in General—IGOR N. ASHESHOV, School of Medicine, University of Western Ontario, London.

A RAPID, three-hours' digestion of meat by papain at 65°C. is described for use as the base of nutrient media. A method of estimation of total oxydizable matter is suggested for standardization of nutrient properties of media in general. The advantages of the method described can be summarized as follows:

1. The preparation is rapid, requiring only three hours for complete digestion at 65°C.
2. The digestion is carried at a temperature which prevents the growth of bacteria during the process.
3. The determination of total organic matter insures more constant composition of media.
4. With an average oxydizable matter of 1.75 per cent in the concentrated broth, and diluting it to 0.75 per cent for use, we obtain from 1 kg. of meat about 10 litres of broth, at a cost of the price of 2 lbs. of meat plus the negligible cost of 10 gm. of papain. No further addition of any nutrient substances is necessary for ordinary purposes.
5. Concentrated broth takes very little room and it is easy to carry a stock of it for a large amount of diluted media for immediate use.
6. The method makes possible variations in the composition of media to suit different purposes. There are three factors affecting the result of digestion which can be varied to alter the final composition: (a) reaction at which

digestion is carried; broth obtained by digestion in acid reaction differs in composition from that obtained in alkaline; (b) temperature affects not only the rate of digestion but alters the composition qualitatively; (c) time of digestion affects it in the same way. By altering these factors considerable variations can be obtained in the final composition of the media, thus making it more suitable for different microorganisms.

Preliminary Observations on the Survival of *S. typhi* in Canadian Cheddar-Type Cheese—L. E. RANTA and C. E. DOLMAN, Connaught Laboratories, Western Division, University of Toronto.

SEVERAL recent outbreaks of typhoid fever in Canada have been attributed to Cheddar-type cheese made from raw milk. In the domestic market such cheese is mostly consumed within a few weeks, or even days, of manufacture, while the present heavy overseas demand has reduced to a minimum the interval between manufacture and consumption of exported cheese. In view of these facts, a thorough investigation into the time of survival of *S. typhi* under various conditions was deemed desirable. A few preliminary findings are now reported. The cheese used throughout the investigation was British Columbia Cheddar ("Stilton style"), purchased in the open market. An overnight broth culture of *S. typhi* (Rawlings rejuvenated strain), diluted with a small amount of nutrient broth to the equivalent of MacFarland's No. 3 turbidity standard, was used as the infecting agent.

Five cc. amounts of the standardized *S. typhi* suspension were intimately mixed with 30 cc. portions of the cheese, which had been previously softened by passage through a fine mincer. The samples of infected cheese were then packed into Petri dishes, which were sealed with parafilm and stored either at room or refrigerator temperature. At intervals, the plates were unsealed, and a sample of cheese removed with a cork borer. The sample was macerated with about twice its volume of sterile saline by means of a glass rod and beads, and single drops of the resulting suspension were spread on plates of various selective media. Endo's medium yielded far greater numbers of *S. typhi* colonies from the cheese suspensions than did either McConkey's, eosin methylene blue, purple lactose, or bismuth sulphite agar media. On three occasions, each involving a separate purchase of cheese, there was remarkable consistency in the survival time at room temperature (68°F.). Sample 1 was positive up to the twenty-sixth day after inoculation, and was negative when tried on the thirty-fifth day; sample 2 was positive up to the twenty-eighth day, and negative on the thirty-second day; and sample 3 was positive up to the twenty-sixth day, and negative on the twenty-eighth day. These preliminary experiments suggest that at room temperature *S. typhi*, intimately admixed with Canadian Cheddar-type cheese, may survive therein, in detectable numbers, for roughly one month. In the refrigerator, survival is more prolonged, one sample being still positive seventeen weeks after inoculation.

A very similar result to the foregoing was obtained in an attempt to determine the survival time of *S. typhi* in cheese whose surface had been con-

taminated by infected whey. A MacFarland No. 3 suspension of *S. typhi* was made in Seitz-filtered whey, and 5 cc. of this was inoculated on to the surface of a piece of cheese cut to fit a Petri dish. The dish was sealed with parafilm, samples being taken at intervals for culture as described above. Samples from a room-temperature cheese were positive up to the twenty-sixth day, but were negative on and after the twenty-ninth day; while similar samples from the refrigerator were still positive on the seventy-fifth day. This finding raised the possibility that survival of *S. typhi* in surface-contaminated cheese might depend upon penetration of the microorganisms into the cheese substance, and the following method was employed to test the extent of such penetration.

Parowax-coated cylinders of cheese were made, about 12 cms. long by 3 cms. in diameter. A thin slice was removed from one end of each cylinder, and a small well, about 0.5 cm. in diameter by 1 cm. deep, bored in the centre of the cut surface of the larger portion. Into this well was pipetted 0.5 cc. of an overnight broth culture of *S. typhi*. The slice was replaced, after its cut surface had been wax-coated, so that any penetration of the cheese by *S. typhi* was necessarily into the larger portion of the cylinder, and the whole cylinder was then re-waxed.

By cutting such cylinders into slices 1 cm. deep, and culturing from the proximal cut surfaces, the rate and extent of penetration of *S. typhi* through the cheese substance could be determined. A pair of cheese cylinders stood on end (one upside down) in the refrigerator, when examined on the seventeenth day after inoculation, showed that *S. typhi* had penetrated 4-5 cms. in both the upright and the inverted cheese cylinders. Gravity apparently has little effect upon the penetration, and capillarity rather than active migration appears chiefly responsible for the process.

The significance of these preliminary observations is briefly discussed in terms of such possible control measures as the exclusive use of pasteurized milk in the manufacture of Canadian Cheddar-type cheese; the storage of cheese prior to consumption; the examination of personnel in cheese factories for exclusion of typhoid carriers; and the routine bacteriological testing of cheese samples prior to release of large consignments for domestic or overseas consumption.

Chemotherapeutic Study of Simple Nitro Compounds—C. SIEBENMANN and R. J. SCHNITZER, Connaught Laboratories, University of Toronto.

IN an attempt to increase the slight antipneumococcic activity of morpholine, a number of N-derivatives of morpholine were prepared. The acetyl, i-amyl and the benzoyl derivatives were found to be inactive. The p-nitrobenzoyl compound of morpholine, however, shows a slight activity upon the pneumococcus in mice and a marked activity against the meningococcus infection.

This observation led to a more systematic study of a number of simple nitrocompounds: 4-nitrophthalic acid, 3-nitrosalicylic and 5-nitrosalicylic

acids show little or no activity on pneumococci and streptococci in mice, while p-nitrobenzoic acid proves to be slightly active against highly virulent strains of both microbes, thus confirming previous observations by Mayer and Oechslin (Arch. Intern. Pharmacodyn, 1939, *62:* 111) and Bauer and Rosenthal (Pub. Health Rep., 1939, *54:* 1317).

In addition to the p-nitrobenzoyl derivative of morpholine (I) the corresponding N-substituted piperidine (II), piperazine (III), dibenzylamine (IV), cyclohexylamine (V), aniline (VI), phenylenediamine (VII), benzidine (VIII) and thujylamine (IX) were prepared as well as the p-nitrobenzoylesters of phenol (X) and cyclohexanol (XI). None of these compounds show more than a trace of activity upon the pneumococcus, while the compounds I and II are slightly active against the streptococcus. The cyclohexanolester (XI) possesses a marked antistreptococcic activity. The compounds I, II and XI are active against the meningococcus infection of mice. In the case of I and XI, this antimeningococcic activity is considerable but not as good as that of sulfanilamide and some other sulphonamides and sulphones.

Subsequent to our first observation of a certain chemotherapeutic activity of the p-nitrobenzoyl morpholine, our attention was drawn to a study of nitrobenzoyl compounds by Mayer and Oechslin. These authors tested the therapeutic activity of a group of different p-nitrobenzoylesters and claim that some of them are more or less active on pneumococci, streptococci or both. They report a marked antipneumococcic and antistreptococcic activity of the cyclohexanolester of p-nitrobenzoic acid. Our own observations, while confirming the antistreptococcic activity of the cyclohexanolester (XI), do not corroborate the marked antipneumococcic activity claimed for this compound by Mayer and Oechslin.

From the experiments with all nitrocompounds examined so far, we can draw the following conclusions concerning the connection of chemical constitution and bactericidal activity.

Definite chemotherapeutic activity depends on the presence of the p-nitrobenzoyl group. The importance of the NO_2 group is indicated by the fact that the NO_2-free compounds are inactive.

A second NO_2 group introduced into the p-nitrobenzoyl rest seems to decrease the chemotherapeutic activity. The 3, 5-dinitrobenzoyl-morpholine, the 3, 5-dinitrobenzoyl-piperidine and the 3, 5-dinitrobenzoyl-cyclohexanol are much less active than the corresponding compounds I, II and XI.

The study of nitrocompounds will be continued in both the chemical and the biological direction.

The Antitoxin Response to Tetanus Toxoid Combined with Typhoid-Paratyphoid A and B Vaccine—L. GREENBERG and J. GIBBARD, Laboratory of Hygiene, Department of Pensions and National Health, Ottawa.

THIS is a report of an experiment designed to determine the effect on the antigenic activity of tetanus toxoid combined with typhoid-paratyphoid

vaccine. Ramon (1) (2) has claimed that such a combination not only does not interfere with the activity of the toxoid but that the reverse is true, in that the vaccine appears to act as an adjuvant, resulting in increased response to the toxoid.

The toxoid plus vaccine mixtures used in the experiment were made by the addition of fluid tetanus toxoid to an equal volume of a vaccine prepared for human use, consisting of 1500 million typhoid, 500 million paratyphoid A, and 500 million paratyphoid B organisms per cubic centimetre. These mixtures were allowed to stand over night in the refrigerator before use.

For purposes of control, an inoculum was prepared consisting of equal parts of the same tetanus toxoid and physiological saline. Thus all animals received the same amount of tetanus toxoid of the same strength.

The following scheme of inoculations and bleedings was followed for each of two groups of 120 guinea-pigs:

(a) All pigs were bled before the first inoculation.

(b) Group I received 0.25 cc. tetanus toxoid plus 0.25 cc. T.A.B. vaccine. Group II received 0.25 cc. tetanus toxoid plus 0.25 cc. saline.

(c) After six weeks all were bled, then Group I received 0.5 cc. of toxoid plus 0.5 cc. of T.A.B. vaccine, and Group II received 0.5 cc. of toxoid plus 0.5 cc. of saline.

(d) Six weeks later (c) was repeated.

(e) Six weeks after (d) all animals were again bled.

The sera from the individual blood samples were each titrated for tetanus antitoxin, using guinea-pigs. All of the 240 pigs showed less than a thousandth of a unit of antitoxin per cc. of blood before injection of the first dose of antigen (table 1). At the fourth bleeding, six weeks after the third dose of antigen, it was found that of those animals which had received toxoid plus T.A.B. vaccine, 24.4 per cent possessed more than two units of antitoxin per cc. of blood, 42.3 per cent between one and two units, and 33.3 per cent between 0.1 and 1 units. During the same interval, those animals receiving toxoid plus saline showed 19 per cent with more than two units of antitoxin per cc. of blood, 45 per cent with between 1.0 and 2 units, and 36 per cent with between 0.1 and 1.0 of a unit.

It is concluded that mixing the antigens in the manner described did not reduce the efficacy of the tetanus toxoid. It is also concluded that the T.A.B. vaccine did not, under the conditions of the test, act as an adjuvant to increase the antigenicity of the tetanus toxoid.

REFERENCES

1. Ramon, G.: Mixed Vaccinations. New Internat. Clin. 1:241-255, 1939.
2. Ramon, G.: L'Anatoxine tetanique et les Nouvelles Methods de lutte contre le tetanos. Bull. Acad. de Med. Paris, 121:609-636, 1939.

THE ANTITOXIN RESPONSE IN GUINEA-PIGS TO INOCULATION WITH TETANUS TOXOID WITH T.A.B. VACCINE AND WITH SALINE

	Tetanus Toxoid plus T.A.B. Vaccine							Tetanus Toxoid plus Saline						
	No. of Guinea Pigs	Units of Antitoxin						No. of Guinea Pigs	Units of Antitoxin					
		>2.0	≥1.0 <2.0	≥0.1 <1.0	≥.01 <0.1	≥.001 <.01	<.001		>2.0	≥1.0 <2.0	≥0.1 <1.0	≥.01 <0.1	≥.001 <.01	<.001
		%	%	%	%	%	%		%	%	%	%	%	%
First titration before inoculation	120	—	—	—	—	—	100	120	—	—	—	—	—	100
Second titration 6 weeks after first inoculation	117	—	—	1.8	48.7	50.5	—	118	—	—	2.5	43.3	54.2	—
Third titration 6 weeks after second inoculation	116	—	56.8	40.7	2.5	—	—	110	—	48.0	42.1	9.9	—	—
Fourth titration 6 weeks after third inoculation	102	24.4	42.3	33.3	—	—	—	105	19.0	45.0	36.0	—	—	—

A New Phage and a Susceptible W Form of *S. typhi* isolated from a Typhoid Fever Case—C. E. DOLMAN, DONNA E. KERR, and DOROTHY E. HELMER, Division of Laboratories, Provincial Board of Health of British Columbia.*

A PHAGE possessing lysogenic powers for many V and W forms of *S. typhi*, but lacking such powers for certain of Craigie's types of V forms, and for their corresponding W forms, was isolated on several occasions by stool culture from a severe case of typhoid fever. By blood culture on the tenth day of illness, and on several subsequent occasions by stool culture, a W form of *S. typhi* was isolated from this case. No V form colonies were at any time found.

The Incidence of Human Trichinosis in Toronto—ELLA KUITUNEN-EKBAUM, Department of Hygiene and Preventive Medicine, University of Toronto.

THERE are no published data on the incidence of human trichinosis in Canada. The data given below are the results of a survey of 420 human diaphragms in Toronto. Of these, 300 were obtained from the Banting Institute, 133 of which were from females and 167 from males. The ages of these people ranged from 15 to 86 years. One hundred and twenty diaphragms were obtained from the Hospital for Sick Children, 51 from females and 69 from males. The ages of the children were up to 16 years. This survey includes two premature infants, 9 infants less than one day old and 66 from one day to one year old.

Six positive cases were found from the autopsies in the Banting Institute. The seventh case was from a premature infant in the Hospital for Sick Children. The latter is of interest as a case of prenatal infection in which the larvae of *Trichinella spiralis* had been carried from the mother to the child. All seven cases of trichinosis found were unrecognized clinically.

According to the results of this survey the incidence of human trichinosis is quite low in Toronto and it is probably no higher in other parts of Canada. Surveys on hog trichinosis carried out by Dr. T. W. M. Cameron, Macdonald College, Quebec, have shown a fairly low incidence throughout Canada. The incidence of trichinosis in man is usually in correlation with that in the hog as the hog is the main source of human infection.

Report of a Case of Dipylidium Caninum in a Child—ELLA KUITUNEN-EKBAUM, Department of Hygiene and Preventive Medicine, University of Toronto.

DIPYLIDIUM CANINUM, a common and widely distributed tapeworm of dogs, cats, and their wild relatives, has been occasionally recorded in man in different countries. Most of these cases have been reported in children, although several cases of adult infections are known.

**This paper will be published in full in the March issue.*

No human cases of *D. caninum* infection have yet been reported in Canada, though the parasite is very common in dogs and cats here.

The present case was brought to my attention by Dr. Patricia Wanning, who sent me a proglottid that had been expelled by a three-year-old child, a clinical patient at the Hospital for Sick Children. According to the information obtained, the child usually passes one or two proglottids in her stool. The child has not yet been treated and, therefore, it is unknown whether she harbours one or more specimens of *Dipylidium caninum*.

Photomicrographic Method of Recording Pathological Diagnoses in Cancer Clinics—JAMES MILLER, Queen's University, Kingston, Ontario.

THE recording of pathological diagnoses in the case histories of patients who come up for advice and treatment to cancer clinics seems a simple enough matter. There is first the report by the pathologist written out in detail and giving the gross and microscopic findings and the category into which the tumour or tissue falls. By way of reference, in a case which is in doubt, there is the microscopic slide or slides to which recourse can be made. This latter may involve some time and trouble. It may mean a trip to the laboratory where a microscope is available. It is much better that the review of the case be carried out in the office where the case histories are filed or at the monthly meeting of the board.

For some time past it has been our practice in the Kingston Cancer Clinic to file along with the case histories micro-photographs of the slides prepared from the biopsies. The procedure is to take a very low power view of the whole biopsy or the most representative portion thereof showing its outstanding features and, in addition, one or more higher power views showing the finer details and the characters of the cell constituents. It may be claimed that this presents no great advance on the mere classification of the tumour in a particular group; that everyone understands what is meant by a melanoma or an epidermoid cancer. But the trouble is that our medical nomenclature is neither universally accepted nor is it stable. Moreover, new terms are being added at frequent intervals which remain for long unfamiliar to the profession at large. What one man understands by a particular name is not by any means what another understands. To take the example of the skin tumours, we may be clear upon the matter of the main categories—papilloma, melanoma, epidermoid carcinoma, but when we attempt to differentiate between the subvarieties especially of the latter two groups we are immediately in difficulties. Terms such as basal cell carcinoma, squamous cell carcinoma, rodent ulcer, not to mention adenocarcinoma of the sweat or sebaceous glands, acanthoma, Bowen's disease, Paget's disease and so on, may mean one thing to one man and quite a different thing to another. If, on the other hand, there is a series of good photo-micrographs of the tumour, much of the difficulty is removed. The clinician who is reviewing the case will say, "Ah, yes. What our pathological colleague really means is what I understand by the term . . ."

The only objections which can be raised to the method are: the time consumed in selecting the areas and taking the photographs, developing and printing them and, secondly, the expense involved. As regards the former objection, if a batch for a week or a month are dealt with together the time consumed is considerably reduced; as regards expense, one high and one low power photomicrograph can be mounted neatly on a cardboard sheet for almost exactly 20 cents, excluding of course the time of the technician.

One more point may be mentioned and that is that in our experience it is by no means infrequent that on looking over the photomicrographs the pathologist observes points not previously seen by him and he may actually alter his diagnosis somewhat in the light of what the photographs reveal.

The Concept of Bacterial Equilibrium and its Possible Application to Soil Problems—A. G. LOCHHEAD, Division of Bacteriology and Dairy Research, Science Service, Department of Agriculture, Ottawa.

INCREASING attention is being given to the subject of the mutual relationships existing between micro-organisms—to those phenomena included in associative or antagonistic action. However, in such natural habitats as water and soil, bacterial equilibrium in the wider sense is regarded as playing an important role, apart from the mutual effects on each other of any two given species.

Water is believed to have an autochthonous bacterial flora, the numbers and kinds of organisms representing not a haphazard assortment or distribution, but rather an equilibrium changing with many influences. In soil an analogous condition exists but to a more pronounced degree. From the purely microbiological point of view, inter-relationships are decidedly more complicated than in a given food product or an infected animal. There is also the distinction between the base from which the soil microbiologist "takes off" as compared with say, the medical or food bacteriologist, the ideal state of whose subject connotes relative freedom from, rather than abundance of, bacteria.

What direct study of microbial equilibrium has been done has been concerned largely with the balance between groups of organisms, e.g. bacteria and protozoa. There is, however, a balance among the bacteria not revealed by purely quantitative methods, but only by a quanti-qualitative analysis. This implies classification, which may be based predominantly on morphological or physiological characteristics.

Recent investigations by the Soils Group of this Division have followed both the morphological and physiological approach, the former adding to our knowledge of soil organisms, and confirming the existence of an indigenous flora, the latter approach being more important from the standpoint of the economy of soils. Utilizing nutritional differences as a basis for grouping soil bacteria has suggested a useful method for following changes in the bacterial equilibrium. The basis for the procedure is the determination of the relative incidence of bacteria belonging to different groups according to their requirements for growth.

Studies on a variety of soils have suggested the value of the method in evaluating soils from the standpoint of crop-producing ability. Moreover, the method has been found capable of yielding suggestive results when applied to specific problems, in which abnormal conditions are apparently not due to the presence of any definite micro-organism. Application of the principle involved suggests that many soil phenomena, at present difficult to account for, will be shown to depend on the equilibrium existing between the many members of the soil's micro-population.

The Toxicity and Trypanocidal Activity of Commercial Neoarsphenamine—C. A. MORRELL and M. G. ALLMARK, Laboratory of Hygiene, Department of Pensions and National Health, Ottawa.

COMMERCIAL neoarsphenamine cannot be regarded as a chemical compound but must be considered as a mixture. It consists of arsphenamine with substituted amino groups; both mono- and di-substituted products being present in various proportions. It also contains impurities such as oxidation products, sulphates, sodium chloride, etc. Chemical analyses do not give a reliable estimate of the toxicity or activity of such a preparation, and biological assay is resorted to for this purpose.

A Canadian Standard for neoarsphenamine, the toxicity and potency of which is known in terms of the International Standard, has been adopted, and methods of assay devised for determining the toxicity and potency of commercial samples as per cent of the standard.

The results of 200 such assays for toxicity and 73 assays for trypanocidal activity are reported. Material from nine manufacturers located in England, France, Germany, the United States and Canada has been tested. There are statistically significant differences among the products both in toxicity and activity. These differences are not, however, really great. The most toxic products were frequently found to be the least active, and the least toxic were usually the most potent.

The value of biological tests in assessing the clinical safety and effectiveness of the drug is discussed.

The Quantitative Determination of Arsenic in Limestone Rocks by a Modification of Morris and Calvery's Technique—J. WYLLIE, Professor of Preventive Medicine, Queen's University, Kingston.

IN the Canadian Public Health Journal for March 1937, a brief account was published of "An Investigation of the Source of Arsenic in a Well Water." The incriminated well is situated on a farm near Madoc, Ontario, and is drilled to a depth of 94 feet in a dolomitic limestone stratum. After numerous filtration experiments the conclusion was reached that the arsenic was not in solution in the well water but in the form of very fine particular suspension.

This result led to an examination of the scale in the farmer's kettle and of pieces of limestone exposed by digging in the vicinity of the well. Chemical tests and microscopic examination of geological sections of the kettle scale and of the natural limestone rock revealed the presence of an iron-arsenic compound.

In July 1937 and again in 1938 the geological formation of the Madoc area was studied in order to ascertain the extent of the dolomitic limestone stratum. Samples of rock were collected from different outcrops on the Hauser farm, on the Ottawa-Sarnia highway in the vicinity of the Hauser and closely adjoining farms and of the local cheese factory. The following table shows the sites of the outcrops and the content of arsenic in seventeen samples examined. The iron-arsenic compound is not distributed evenly throughout the stratum. In the chemical examination of individual outcrop samples, however, the rock was thoroughly ground to a fine powder before the tests were applied.

RESULTS OF QUANTITATIVE ESTIMATION OF ARSENIC IN DOLOMITIC LIMESTONE OUTCROPS

	Rock Sample	Arsenic as As_2O_3 in parts per million
I	Outcrop where Ottawa-Sarnia highway curves sharply to N.E., 1 mile east of Madoc	0.726
II	Outcrop in field 60 feet S.S.W. of Hauser barn	1.981
III	Outcrop in yard 80 feet S.W. of Hauser barn	5.348
IV	Outcrop outside pumphouse	0.502
V	Outcrop from a cut on Ottawa-Sarnia highway 400 feet east of Hauser farm-house	1.928
VI	Outcrop from a cut on Ottawa-Sarnia highway at concession corner west of T. Feeney Sr.'s house	0.621
VII	Outcrop south of cheese factory	1.426
VIII	Outcrop 1 mile south of cheese factory, adjacent to Hauser farm	5.757
IX	Outcrop 100 feet S.E. of Barn on Hauser Farm	4.344
X_A	Rock sample unearthed near piggery	5.057
X_B	" " " " "	6.325
X_C	" " " " "	0.924
Field Sample	I Rock picked up in ploughed field	0.660
Field Sample	II Rock picked up in ploughed field	0.845
Field Sample	III Rock picked up in ploughed field	0.885
Rock sample from Highway Cut III		5.651
Rock sample from Highway Cut IV		10.735

A modification of Morris and Calvery's technique (Ind. & Eng. Chem., Anal. Edit., 1937, *9*: 447), originally devised for the estimation of arsenic in biological materials, has been found suitable for the quantitative determination of small amounts of arsenic in these rocks. Briefly the steps in the technique are: (a) preparation of the outcrop sample of rock; (b) extraction of the iron-arsenic compound in acid solution; (c) exsiccation of the rock residue at 50°C. approximately; (d) conversion of the arsenic compound, in residue and extract combined, into arsine, with immediate decomposition into metallic arsenic, and (e) development of a blue-coloured arsenic compound in acid-molybdate solution with subsequent measurement in a photo-electric colorimeter.

Isolation of Poliomyelitis Virus from Two Cases: Laboratory Findings—JAMES CRAIGIE, School of Hygiene and Connaught Laboratories, University of Toronto.

THIS report deals with the isolation of poliomyelitis virus from the stools of two cases. The strains of virus were recovered by intraperitoneal inoculation of processed stool extracts. The specimen yielding virus from the abortive case was obtained one month after the illness and virus was recovered from the specimen after it had been stored as long as six months in the cold room.

Report of a Trial of a New Schick Toxin—G. D. W. CAMERON and J. GIBBARD, Laboratory of Hygiene, Department of Pensions and National Health, Ottawa.

THIS is a report of a comparison of the results of using Taylor and Moloney's "fresh" Schick toxin (1) and the "old" or League of Nations standard Schick toxin, with the Moloney test as a control. Each individual was given the "fresh" Schick toxin in one arm and the "old" toxin and control in the other arm. Readings were obtained on 136 people, previously bled for the titration of the diphtheria antitoxin level. They ranged in age from 18 to 61 years with four-fifths in the 20- to 49-year group. Immunity was not more apparent in one age group than in another.

Sixty-eight, or exactly half, were found to possess less than 1/500 of a unit of diphtheria antitoxin per cubic centimeter of blood; 63 of these were frankly Schick positive when both "fresh" and "old" Schick toxin reactions were read. Of the remaining five, reading the "fresh" toxin reactions plus controls, three were positive, one was a doubtful positive and one was definitely negative; reading the "old" Schick toxin reactions plus controls, one was positive with slight reaction to the control, two gave doubtful reactions, and two were definitely negative.

The remaining 68 persons possessed 1/500 of a unit or more per cc. of blood; 31 of these were reactors. Among the latter, reading the "fresh" toxin reactions plus controls, 24 or 77 per cent could be interpreted, whereas, if reading were confined to the "old" toxin reactions plus controls, only 15 or 48 per cent could be interpreted.

It is concluded that the new Schick toxin prepared from "fresh" diphtheria toxin gave a higher percentage of interpretable reactions than that prepared from matured or "old" toxin.

REFERENCE

(1) Taylor, E. M., and Moloney, P.J.: J. Immunol., 1939, *37*, 223.

Single Colony Isolation of Anaerobes*—L. GREENBERG, Laboratory of Hygiene, Department of Pensions and National Health, Ottawa.

IN dealing with anaerobes, it is common experience that pure cultures are difficult to obtain. The most reliable methods of obtaining pure cultures of anaerobes are those employing single cell techniques, which are difficult, tedious, and can be carried out only by an expert who has devoted considerable time to mastering the details.

A procedure, based on the work of Ward and Rudd's technique for the separation of single colonies of streptococci (1), has been developed. This method is simple and quick; no special technique or apparatus is required. It is as follows:

PROCEDURE:

1. *Medium:*

Proteose peptone	10.0 g.m.
Tryptone	10.0 "
Sod. thioglycollate	1.0 "
Agar	3.0 "
Distilled water	1000.0 cc.

Adjust the reaction to pH 7.4, dispense in tubes in 10 cc. amounts and autoclave at 15 pounds for 20 minutes.

2. *Technique:*

Using a 1-mm. platinum loop, one loopful of an 18-24 hour broth culture is touched gently on the inside of the culture tube, so that only the barest film of culture remains on the loop. This inoculum is then transferred to a tube containing 5 cc. of sterile broth (type of broth is immaterial) and thoroughly mixed. The loop is flamed and a loopful of the inoculated broth is transferred to a tube of the semi-solid medium, which has been heated in boiling water to displace oxygen and cooled to 45°C. immediately before use. The tube should then be rotated to distribute the organisms evenly. It is then placed in the incubator under ordinary aerobic conditions.

The incubation period will vary with the organism under study. For *Cl. welchii* the best incubation period was found to be overnight (16-18 hours) at 37°C. For all other anaerobes studied it was found best to leave the cultures at room temperature (approximately 22°C.) overnight and place in the 37°C. incubator the following morning. The development of colonies is then watched closely (up to 10 hours), the cultures being removed from the incubator when the colonies have reached a suitable size. Prolonged incubation at 37°C. may result in confluent growth, making it impossible to obtain single colonies.

In the case of mixtures of anaerobes and aerobes it has been found possible to eliminate the latter by (a) preliminary anaerobic cultivation of the mixture in fluid medium, and (b) by dilution of the resultant growth to the point

**Presented at the meeting as a demonstration.*

where aerobic organisms are eliminated in the majority of tubes planted. The following procedure for dealing with contaminated material has been tried and has proved successful:—

1. The product on test, or a representative portion, is inoculated into 250 cc. of Brewer's thioglycollate medium (without dextrose) which has been previously heated in boiling water, and cooled to 45°C.

2. The inoculated flask is incubated anaerobically in a McIntosh and Fildes' jar for 24 hours at 37°C.

3. From this, at least 20 tubes, containing semi-solid medium, are inoculated using the previously described method. These are incubated aerobically.

Using the above technique and medium, separate colonies were successfully obtained from each of the following cultures of anaerobes: *Cl. botulinum, Cl. chauvei, Cl. fallax, Cl. histolyticum, Cl. novyi, Cl. putrificum, Cl. sordelli, Cl. sporogenes, Cl. tertium, Cl. tetani, Cl. tetanoides, Vibrion septique, and Cl. welchii.*

REFERENCE

(1) Ward, H. K., and Rudd, G. V.: Studies on Haemolytic Streptococci from Human Sources. Australian J. Exper. Biol. & M.Sc., 1939, *17:* 77-79.

Studies on H. Pertussis—NELLES SILVERTHORNE, Connaught Laboratories, University of Toronto, and Hospital for Sick Children, Toronto.

IN this paper the author believes that the value of an active immunity agent against whooping cough is best tested clinically by determining the number of cases of the disease that occur in a control and vaccinated series who have been exposed to positive "cough plate" patients. Fresh strain phase I pertussis vaccines have been shown to immunize children by thirteen different workers compared to failure to do so by two.

Mice are completely protected against a fatal septicaemia produced by *H. pertussis* and mucin intraperitoneally when previously inoculated with phase I fresh strain vaccines. Other products do not protect.

Minimal doses of pertussis vaccine (0.3 cc. to 0.4 cc., Gates 2, approximately 15,000 to 20,000 million per cc.) completely protect mice against a fatal septicaemia produced by the intraperitoneal injection of phase I *H. pertussis* and mucin.

Agglutinins and mouse-protecting antibodies have been developed in the blood of vaccinated children. Precipitins have not been detected. Bactericidal antibodies are questionably present.

An immune rabbit serum has been developed showing high-titre agglutinins and precipitins as well as mouse-protecting antibodies. This serum does not neutralize the dermonecrosis produced in the skin of rabbits as a result of "pertussis toxic material".

Preliminary studies on the use of immune rabbit serum in the immediate prevention of whooping cough in children after contact suggest that more extensive studies should be undertaken.

EDITORIAL SECTION

THE PRESENT STATUS OF OUR KNOWLEDGE OF INFLUENZA VIRUS

IN this issue there appears a paper by Dr. Ronald Hare reviewing the present status of the virus of influenza. The problem of influenza came measurably nearer solution with the discovery of a virus in 1933, which subsequent work has shown to be the probable cause of many cases of the disease. This virus, now known as influenza A, has appeared in some quarter of the globe every year since that time. For a time, particularly after the experience of both British and American workers in the epidemic of 1937, it seemed highly probable that this was the only virus to be found in epidemic influenza, but recent epidemics have not borne this out, and in some of them a second virus, influenza B, has been isolated. The latter is immunologically quite distinct from influenza A, but nevertheless produces in human beings an infection clinically identical with that caused by the virus first discovered.

Dr. Hare has drawn attention to the probability that other agents may produce infections clinically identical with epidemic influenza. In recent studies, bacteria, rickettsiae and viruses have been related to such infections. The clinical picture in these infections may resemble that of epidemic influenza so closely that on clinical grounds alone it is next to impossible to make a differential diagnosis. This is not surprising since the diagnosis of influenza rests largely on the presence of a febrile disturbance with rather vague symptoms in the respiratory tract. If a number of patients are ill at the same time, the expression "epidemic influenza" is used, but it is highly probable that it is the epidemicity rather than the clinical findings which plays a principal part in formulating the diagnosis. If the outbreak is sporadic, the term "influenza" may still be used, but in this instance because of the lack of any more accurate designation for the particular illness in question. It is debatable, therefore, whether or not we should stop designating cases as influenza when the diagnosis is based on clinical signs only, and either invent a new name or use the old army term "pyrexia of unknown origin" (P.U.O.), which at least has the merit of accuracy.

This is not of mere academic interest. It is actually of great importance, for even if we possess an immunizing agent for one of the influenza viruses and immunize a group of persons, we shall inevitably be confronted with a great many

infections in the immunized group which can only be diagnosed as "influenza". Some will be cases of common cold which recent work has shown to be quite distinct from influenza proper, although when severe, a common cold may mimic influenza very closely. And there will be other infections. Some of these may be due to one of the other influenza viruses for which the immunizing antigen has not produced immunity, but others may be due to one or other of the agents, known and unknown, which may also produce an influenza-like syndrome. This question has assumed great importance recently in view of the recent discoveries at the Rockefeller Institute which render it highly probable that we do possess an immunizing agent for one of the influenza viruses, now designated as influenza A. Whether it is possible to make an antigen of similar value to protect against the other causative agents concerning which there is now some information, is for the future to show. There is insufficient knowledge at present to warrant the attempted immunization of the general population. During a severe epidemic, however, it might be wise to modify this attitude, particularly for key personnel.

THE CONTRIBUTION OF THE CANADIAN HEALTH CONSERVATION CONTESTS TO THE MAINTENANCE OF PUBLIC HEALTH IN WARTIME

IN the present trying times many medical health officers and health departments in Canada have additional duties to perform, and some of the staff may be absent on military duties. Nevertheless they are trying to maintain the high level of public health services. It is essential to prevent any retrogression or curtailment of health activities. Not only must the armed forces of Canada be kept physically fit, but the health of the rank and file of workers standing back of them must be safeguarded. At a time when the nation's resources are being mobilized to the full, health conservation is of first importance.

During the past four years medical officers of health in charge of rural health districts have learned of the value of the Canadian Health Conservation Contest. Basically, the plan affords the health officer an opportunity to enlist the co-operation of the leading laymen of the community who constitute the contest committee; and to survey, by means of the fact-finding schedule, the accomplishments and needs of the department. Participation in these contests was first made possible for rural units in Canada in 1937 through the co-operation of the American Public Health Association and with the financial support of the W. K. Kellogg Foundation. The splendid response and the very evident value of the effort led to the desire on the part of the Canadian Public Health Association to extend the contest to urban centres. Again through the co-operation of the American Public Health Association and with the support of the Metropolitan Life Insurance Company, the first urban health contest is being held this year.

The preparation of the schedule form, presenting the health activities of the past year, is well worth while. The schedules, when properly filled out, are documents of much value both to the health department and to the local community. They are also valuable historical records and measuring lines to evaluate

the work of one year with that of other years, or with that of the same year in other places.

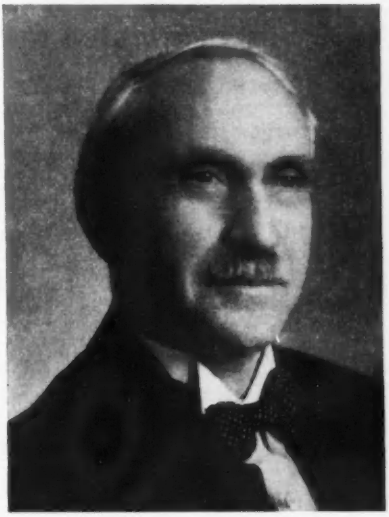
One of the chief benefits which accrue to a participating unit is the opportunity which is presented for awakening interest in public health projects. Desirable advances have often been planned, but never made, in the absence of public support. The contests can give, and often have given, the necessary stimulus. The contests also furnish a large measure of fusion in bringing together different groups in a community and in getting them to focus attention on a needed project. Participation has demonstrated to communities and health departments the value of records, so that provision has been made for recording the health items and activities which are worthy of taking into account. The preservation of such records has been most useful and time-saving to a health department when requests come for information, and has frequently been of direct service to the community as a whole.

JOHN W. S. McCULLOUGH, M.D., C.M., D.P.H.

An Appreciation

By FRED ADAMS, M.B., D.P.H.

THE passing of Dr. John W. S. McCullough, who died at his home in Toronto on January 5, 1941, will have been noted and mourned more widely perhaps than that of any other doctor in Canada. When the word of his death came, I am sure that, like myself, a thousand Ontario health officers in cities and towns, in rural parts of old Ontario and in the wide stretches of new Ontario, paused in their daily rounds to pay him tribute. They had known him for a full quarter century as the executive officer of the Ontario Department of Health. In those twenty-five years the present public health machine was built, and he more than anyone else had the building of it.



Dr. McCullough was born on January 25, 1868, in Peterborough County. He was educated at Owen Sound Collegiate, Trinity Medical School, and the University of Toronto. Following post-graduate study in New York and London, he began medical practice in 1890 at Alliston, Ontario. His public service began early. He served first as a member of the school board and later, from 1900 to 1902, was mayor of Alliston. In 1906 he was appointed a member of the Provincial Board of Health. In 1910 he left his country practice to become Secretary of the Provincial Board and Deputy Registrar General for the Province.

And so began a quarter century of the very finest public service. Public health was new in those days. The quarter century ahead was to be a period of tremendous progress and Dr. McCullough was to contribute immeasurably to it. The record of his contributions reads like an account of public health progress in the first thirty years of the twentieth century.

He was responsible for the consolidation of the Public Health Act in 1912. Based on its English prede-

cessor, it has proved to be a sound Act and has served as a model for similar legislation throughout Canada.

In 1890 the Provincial Board of Health had established at Toronto the first public health laboratory in North America. A branch laboratory at Kingston followed in 1904. Dr. McCullough extended the service in two ways, by enlarging the range of laboratory examinations and by establishing branch laboratories across the province. Laboratories were opened at Toronto, Kingston, London, Peterborough, Ottawa, North Bay, Sault Ste. Marie, and Fort William. Annual laboratory examinations increased from a few thousands to more than half a million. Probably no more complete laboratory service is to be found anywhere.

Ontario, in 1916, was the first province to undertake the free distribution of essential biological products. The August, 1940, issue of the CANADIAN PUBLIC HEALTH JOURNAL contains one of the last articles which Dr McCullough wrote. Its purpose was to voice an appreciation of Dr. J. G. FitzGerald, who established the Connaught Laboratories, where the diphtheria antitoxin distributed free by the province was made. The article gives a fascinating account of the developments that accompanied what was then an experiment. This appreciation of Dr. FitzGerald not only gives a first-hand account of an important event but reveals the quality of Dr. McCullough himself. The free distribution of diphtheria antitoxin was followed by the distribution of tetanus antitoxin, antimeningitis serum, scarlet fever antitoxin, smallpox vaccine, typhoid vaccine, and ten or fifteen other biological products, and later by the free distribution of Insulin to those unable to pay.

Tuberculosis was a great problem. At the beginning of the century the annual death rate from the disease in Ontario was 200 per 100,000 population. Some progress had been made in the cities: sanatoria had been built, clinics had been established, and contacts were being followed up. The rural and semi-rural districts, however, lagged behind. To deal with the situation, travelling tuberculosis clinics were established, to bring to small towns and rural districts the advantages of X-ray and expert diagnosis.

The first Great War focussed public attention on the venereal-disease problem. With Federal assistance, all the Provincial Health Departments took measures to deal with it. In Ontario the Venereal Disease Prevention Act was passed. Nineteen treatment clinics were established in the larger centres and in the smaller places arrangements made for treatment of patients with government assistance. The provisions for venereal-disease control in Ontario have proved comparable to the best on this continent and this oldest of public health problems would appear to be on the way to solution.

Maternal and infant death rates were unsatisfactory. New Zealand had taught the world how to deal with these problems. Ontario established a Division of Maternal and Child Hygiene whose objective was to increase local interest in a well-rounded program to protect both mother and child. The government prepared, for free distribution, an excellent baby book, and assisted local efforts along the lines of demonstration and education.

Early in Dr. McCullough's administration a Division of Sanitary engineering was created. Typhoid fever was rampant in the province. Many public water supplies were unsafe. Sewage disposal was casual. The division was concerned with water supplies, sewage disposal, pasteurization of milk, and all other matters into which sanitary engineering enters in an important way. Typhoid fever might be taken as the test of the work of the division; once so common, it is now a rare disease in Ontario.

Increased industrialization brought its problems and a Division of Industrial Hygiene was organized to deal with them. It has done fine service, particularly in the control of silicosis

consequent on hard-rock mining in Northern Ontario.

In 1931 the Government appointed a Royal Cancer Commission, which carried on investigations in the United States and Great Britain and on the Continent. Dr. McCullough acted as secretary. Following the recommendations of the commission, diagnostic and treatment centres were established with government assistance at Toronto, Kingston, and London, and later at Hamilton, Ottawa, and Windsor.

Dr. McCullough saw his chief ambition realized before he retired in 1935. The Ontario Department of Health stands in the front rank among government health organizations, a fine monument to his administrative and creative talents. On the human side Dr. McCullough saw results that must have given him the most abiding satisfaction: the general death rate reduced a quarter, ten years added to the span of the average Ontario life, typhoid reduced to the status of a rare disease, tuberculosis displaced from first place to tenth as a cause of death, diphtheria deaths cut from 1200 a year to 7, silicosis brought under control in the north, the deaths among babies reduced two-thirds.

Obviously Dr. McCullough did not accomplish all this single-handed. He had able assistants, but these men were his selections and his was the guiding hand. A man, too, is part of the times in which he lives. In the days of Dr. McCullough's stewardship there were four great figures in public health in Ontario: Dr. McCullough at the Parliament Buildings; Dr. John A. Amyot, at first in charge of the laboratories under Dr. McCullough and later Deputy Minister of Health at Ottawa; Dr. J. G. FitzGerald in the Connaught Laboratories and the School of Hygiene at the University of Toronto; and Dr. Charles Hastings at the City Hall in Toronto. All have passed from this present scene, with Dr. McCullough the last to go. I doubt whether my generation will see again in quantity and quality such leadership as we had from these four pioneers in public health.

My own friendship with Dr. McCullough goes back to 1910. It became particularly close after 1919, when I went to Windsor to take over the direction of a joint Board of Health serving Windsor and five other municipalities. He was greatly interested in that experiment and I consulted him often. More and more I became impressed with his sound judgment and progressive outlook. He kept abreast of scientific discovery, sorted out what was good and gave it effect in Ontario. He had his cronies among the health officers and I was proud to be numbered in that group. He was not above advice from either lay or professional sources. In scientific and research matters he turned to Dr. FitzGerald. He had been a country doctor and had the ability to meet all kinds of people on their own ground. More than anyone else I have known, he could "walk with kings nor lose the common touch."

There was nothing parochial about him. He was one of the Canadian representatives on the International Joint Commission which investigated the pollution of boundary waters (Canada and the U.S.A.) about 1912. He interested himself in the affairs of the Canadian and American Public Health Associations and served his country in the first Great War.

His relaxation was golf. I played often with him at Mississauga. Our golf games got to be something of an institution, whenever I was in Toronto in the summer. The nineteenth hole was tea, toast and jam with the valley of the Credit for background. I have no pleasanter recollection.

Dr. McCullough's death is a national loss. The sympathy of many hundreds of health officers goes to his family and we join his many other friends in deploring the passing of a kind friend, a shrewd counsellor, and a wise leader.

CURRENT HEALTH LITERATURE

These abstracts are intended to direct attention to articles that have appeared in other journals during the past month. Any of the journals referred to may be borrowed for three days or longer if desired. Address requests to the secretary of the Editorial Board.

A New Complex Influenza Vaccine

HORSFALL and Lennette of the Rockfeller Foundation recently described a new vaccine against influenza which they apparently discovered by chance and which is of great interest. In order to prevent the spread of distemper among normal ferrets in their animal colony a formalized distemper vaccine was injected subcutaneously into all the normal ferrets. These animals were used shortly after this for experiments with influenza virus and were found resistant to three antigenically different strains. Subsequently it was found that a formalized vaccine containing mixtures of human influenza virus and canine distemper virus protected ferrets against both diseases. The immunity developed operates against all known human strains of the virus and is more complete and of wider valency than that produced by influenza virus alone. This vaccine is at present undergoing extensive human trial.

Editorial, J.A.M.A., 1941, *116:* 142.

The Influence of Dietary Protein on the Toxicity of Sulphanilamide

THE work reported herein was undertaken in an effort to find a means of overcoming the toxicity of sulphanilamide and was suggested by the fact that the chronic toxicity of ingested selenium was greatly influenced by dietary protein. Experiments were carried out by feeding groups of rats certain measured diets and then administering sulphanilamide intragastrically for a prolonged period. During the course of the experiments records were kept of deaths and survivals and careful blood studies were made.

The results showed a greater mortality among animals on a 7 per cent protein diet than among those receiving 30 per cent protein. There was also a high incidence of anaemia in groups on a low protein diet and none in the group receiving 30 per cent protein. The concentration of blood sulphanilamide was somewhat higher in the low protein diet animals, suggesting greater retention, and possibly accounting for the greater toxicity.

M. I. Smith, R. D. Lillie, and E. F. Stohlman, U.S. Pub. Health Rep., 1941, *56:* 24.

Tuberculin Testing in Chicago Schools

THIS report is of particular interest to those in charge of school medical services and to tuberculosis workers. The materials and X-ray equipment used in the survey are described, together with the results obtained. Of the 167,435 school children tuberculin tested, 27,401 or 16.37 per cent were positive and among these 586 new cases were found by X-ray. Analysis of the results also gives interesting information regarding age, sex, and race incidence, severity of reactions, type of disease and type of school, and the problem of the follow-up. In the latter connection it was found that in 90 per cent of the homes of positive reactors the source of infection was already under supervision. An attempt was made to analyse the cost of the survey and an approximate figure of $450.00 per new case was arrived at. For this reason it is felt that case-finding limited to the school age groups is economically unsound and should be replaced by miniature X-ray film surveys in areas of excessive tuberculosis mortality.

Frederick Tice, Am. Rev. Tuberc., 1941, *43:* 96.

Treatment of Pneumonia with Sulphapyridine and Serum

THE treatment of a series of 217 cases of pneumonia is reported in this article. Sulphapyridine was administered to 197 cases, 47 of which were type I, 112 type II, and 38

type III. The other twenty cases, all type II, were given sulphapyridine followed by type II serum. All twenty cases in this latter group showed a striking clinical improvement within twelve hours and the termination of the illness more closely resembled the natural crisis than when sulphapyridine was used alone. One death, due to pericarditis, occurred in this group, giving a case fatality rate of 5 per cent while the rate for the drug-treated type II group was 12.5 per cent. Type III, as usual, proved the most virulent, with a case fatality rate of 29 per cent. Increasing age and the presence of bacteraemia exerted important adverse influences on prognosis. That further trials of combined serum and drug therapy are justified is apparent.

Thomas Anderson and John G. Cairns, The Lancet, Oct. 12, 1940, p. 449.

Colds and Vaccines

IN this article the considerations that weigh for or against the prophylactic usefulness of stock cold vaccines are intelligently analysed. It is pointed out that while colds are currently believed to be of virus origin, the possibility that some may be strictly bacterial in origin still remains and the occurrence of bacterial complications following virus colds is well recognized. There would therefore appear to be some justification for the use of the stock vaccine but against this must be balanced the fact of type specificity, particularly in the case of pneumococcal and streptococcal immunity. The effect of at least some elements of the stock anticatarrhal vaccines would therefore appear to be limited to a stimulation of group specific antibody or of the antibody mechanism nonspecifically, enabling secondary bacterial infections to be warded off or limited. Controlled experiments have usually shown no benefit to the vaccinated group. However, the experience of a large insurance company was that, while the number of colds in vaccinated and unvaccinated persons was the same, the former group returned to work after their colds several days sooner than the others, on the average.

The Lancet, Sept. 28, 1940, p. 397.

Inoculation and Immunity Experiments on Calves with the Vole Strain of Acid-Fast Bacillus

GUINEA pigs and calves were used in studies of the pathogenicity and immunizing power of the acid-fast bacillus which causes epizootics in field voles. In guinea pigs receiving small doses of this organism intraperitoneally or large doses subcutaneously lesions of a retrogressive nature develop which eventually heal completely. Large doses intraperitoneally, however, produce generalized fatal disease which resembles tuberculosis macroscopically and microscopically. A group of guinea pigs was vaccinated with this organism subcutaneously over a period of two months and then four weeks later was injected with virulent bovine tubercle bacilli along with a number of controls. The vaccinated guinea pigs were found to be more resistant to tuberculosis than the controls as represented by an appreciable delay in development of the disease.

In calves the vole acid-fast bacillus produced a tuberculous reaction, chronic in nature, which tended to disappear without leaving many traces. Vaccinated and control calves were tested for resistance to tuberculosis by the oral administration of virulent bovine tubercle bacilli. At autopsy the controls showed typical tuberculosis while five of the vaccinated calves had only trivial lesions and four no macroscopic lesions. Glands from these animals were emulsified and injected into guinea pigs. In some instances tubercle bacilli were recovered, though evidently scanty in the glands, but glands from two of the calves failed to produce tuberculosis in the guinea pigs. Calves injected with the vole strain reacted very positively to tuberculin.

A. Stanley Griffith and T. Dalling, J. Hyg., 1940, *40*: 673.

INDUSTRIAL HYGIENE ABSTRACTS

An Analysis of 1000 Deaths among Railroad Employees Subject to Periodic Examination

IN this paper the author discusses the records showing the causes of death of 1000 railroad employees (New York Central System). Records of all deaths among employees subject to periodic examination have been kept since the spring of 1937.

Difficulty was experienced in compiling the records owing to trouble in getting accurate cause of death. This information was generally obtained from the superintendent's office but the author hopes that eventually they may ascertain the cause from the death certificates. In the list included in this article the causes were obtained by a comparison of the report received from the superintendent with the records of the surgeons of the railroad system. As far as possible, the actual disease causing death is named.

The analyses of deaths included the following observations: One hundred and eight employees were fatally injured but only 36 of them while in line of duty. Four were drowned and 21 committed suicide. The suicides may have been partly due to the unstable business conditions during those years. The average age at death for the entire group was 55.1 while for engineers and firemen (284 out of the 1000) it was 55.38. The figures for vascular disease did not bear out the claim that heart disease is an occupational disease of engineers; 47.5 per cent of the deaths were from vascular disease of all kinds. This figure does not exceed that of any group whose average age is 55.1. The percentage of coronary deaths was almost the same among engineers as among those not engineers.

George P. Myers. Indust. Med., October, 1940, 509.

Simple Ways of Reducing Fatigue

THE urgent demand for immediately increased war production has necessitated changes in standards of living which will inevitably last for longer than the war. In England every effort is being made to ensure that good health shall not be sacrifised to good output figures. Those who demand such changes as increased hours of work from the workers, realize that it is their responsibility to provide the optimum conditions for the workers. From the beginning of the war, the Minister of Labour has sought advice from leading authorities in industrial fatigue. This advice is receiving active support of the management of the larger industrial establishments but its practical value depends upon the co-operation of the workers. Their responsibility and their duty lie in recognizing means of avoiding fatigue during work and in making the most profitable use of their rest periods.

Much of the fatigue common to industrial workers is due to fixation of large parts of the body. The posture of the modern workman is unchanged for long periods of time, resulting in physical exhaustion.

In this article suggestions for retarding and reducing fatigue are outlined and illustrated. It is suggested that welfare workers place leaflets with such information in canteens, factory common-rooms, etc., as a constant reminder to the workers of the necessity of such measures.

F. W. Hornibrook. Indust. Welfare, September, 1940, 287.